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DECORTICATION OF THE NONEXPANDABLE POSTPNEUMOTHORAX TUBERCULOUS LUNG

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EXPERIENCE of army surgeons during World War II with the development of pulmonary decortication in the treatment of fibrosing traumatic hemothorax, both with and without infection, has awakened interest in the possible applicability of this valuable procedure to some of the problems still unsolved in the surgical treatment of pulmonary tuberculosis.

Sampson and Burford[1] in their recent article on "Total Pulmonary Decortication," speculated on the possible applicability of early decortication in the treatment of acute or subacute total tuberculous empyema. There are sporadic references in the older literature to attempts at decortication in the treatment of tuberculous empyema. Apparently the failure of the then used techniques, plus the hazards and complications have prevented this procedure from becoming a popular therapeutic measure for this indication.

At the Twenty-sixth Annual Meeting of the American Association for Thoracic Surgery, May, 1946, a paper was read by Dr. Frazer B. Gurd on "Decortication in Tuberculous Empyema." In the discussion of that paper one of us (D. M.) presented a brief résumé of one of the case reports which make up the basis of this present paper.

We wish to present the case material herewith as the early results of an attempt to apply the basic principles of pulmonary decortication to the problem of the postpneumothorax nonexpandable tuberculous lung. We have not found reference in the literature to the use of decortication for this indication up to the time of preparation of this paper.

Fully realizing the chronicity of the nature of tuberculosis, the proneness to recurrence and exacerbation, and the long period of postoperative observa-

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tion necessary to establish the validity of conclusions on the efficacy of any therapeutic procedure used in the treatment of pulmonary tuberculosis, we recognize that the period of postoperative observation in our present case material is much too short. We therefore wish to present it only as a preliminary report to be followed, we hope, by a more thorough analysis when our series of cases has increased, and an adequate postoperative observation period has passed.

All who have had experience with pneumothorax therapy for pulmonary tuberculosis encounter cases in which, after a successful pneumothorax has been maintained for the optimal time, the lung will not re-expand after pneumothorax refills have been abandoned. The percentage incidence of this complication is probably not large. However, reliable figures as to its frequency seem to be lacking in the literature. Farber[2] stated that in a limited experience roughly 5 per cent of cases of therapeutic pneumothorax terminate in unexpandable lungs. He reported the occurrence of 27 cases in 318 patients receiving pneumothorax treatment from July, 1939, to July, 1941, and emphasized its significance as a clinical problem. John Alexander[6] discussed it as an indication for thoracoplasty. He gave as causes of the failure of the lung to expand:

1. Extensive destruction and fibrous contraction of a greater part of the lung.
2. A rigid, thick visceral pleural that is often the result of a pleural effusion or empyema.
3. Stenosis of the large bronchi with resultant atelectasis.

The inability of a lung to expand after the abandonment of pneumothorax refills usually results in (1) highly negative intrapleural pressures due to absorption of some of the pneumothorax air. Clinically this may produce a disagreeable sensation of tension, dyspnea, and sometimes a pleural effusion, which recurs frequently following repeated thoracenteses (*Collapsus imperatifs of Veran*). (2) The drawing toward the chest wall of the collapsed lung together with the mediastinum and its structures, the trachea, heart, and great vessels. As a consequence the opposite lung may become overdistended and emphysematous, and latent tuberculous foci may be opened. The vital capacity may be actually decreased. Farber[2, 3, 4] pointed out the dangers inherent in the "dead pleural space" as "chiefly empyema, tuberculous or mixed infection, and broncho-pleural fistula."

The remedial measures applicable to this situation have in the past been: (a) Indefinite continuation of therapeutic pneumothorax, (b) substitution of oleothorax for pneumothorax, (c) thoracoplasty.

In relation to the causes of nonexpandibility enumerated by Alexander[6]: the first, extensive parenchymal lung destruction and fibrosis and the third, bronchostenosis and atelectasis, would still call for thoracoplasty. Both must be given adequate consideration in planning any therapeutic approach. Judgment as to the extent of lung destruction and fibrosis will depend in large measure on the appearance of the entire series of x-ray films and the appearance of the collapsed lung at the time re-expansion is contemplated. Bronchostenosis as a cause of nonexpandability can be ruled out by bronchoscopy. Very often bronchoscopy of a collapsed lung will reveal a slit-like narrowing of the main or

branch bronchi. This condition is mechanical and is due to compression or angulation of the bronchus and it is not a hindrance to expansion. It should not be confused with concentric cicatricial stenosis due to previous inflammatory pathology of the bronchial mucosa, which may be a definite hindrance to re-expansion.

It is with the second cause of nonexpandability cited by Alexander[6] that we are concerned here, that is, the so-called rigid, thick visceral pleura. Farber,[2, 3, 4] in summarizing the causes of nonexpandability stated, "The important factors in preventing re-expansion seem to be visceral pleural thickening (pachy-pleuritis) and extensive post-tuberculosis parenchymal fibrosis." We believe that this concept of a pachy-pleuritis, or thickening and fibrosis of the visceral pleura, is in at least a great many of these cases a pathologically erroneous one. It had often been noted at postmortem examination of patients dying while under pneumothorax treatments that the hyalinized fibrous envelope which encased the collapsed lung could be readily peeled away from the visceral pleura, leaving the latter intact and fairly normal in appearance except for small areas here and there, especially overlying the most diseased portion of the lung parenchyma, where the underlying parenchymal inflammation had penetrated the visceral pleura and caused it to become intimately adherent to the envelope. With the advent of resection surgery for pulmonary tuberculosis, this condition was again noted in mobilizing a lung or lobe prior to resection, if an unsatisfactory pneumothorax of some duration preceded the indication for resection.

The pathology then is not always inflammatory thickening of the visceral pleura itself, although that undoubtedly does sometimes occur particularly if the pneumothorax has been complicated by a true bacillary tuberculous effusion. Not uncommonly it consists of the laying down of a hyalinized fibrous layer on the pleura (Figs. 9 and 10) to which it is not intimately adherent in large part, but attached by a thin areolar vascularized layer (Figs. 10 and 11) which may be easily separated by blunt dissection, leaving behind an intact and, for the most part, fairly normal and expandable visceral pleura. This fibrous envelope (Figs. 5, 9, 10, and 14) may vary from a thin transparent pliofilm-like layer to a thickness of 1/4 inch or more, but regardless of thickness the character of the loose areolar layer (Figs. 10 and 11) approximating the pleura is the same.

This fibrous envelope is in all respects similar to that described by Delorme,[7, 8, 9] Lilienthal,[10, 11] and others in the older literature as being the underlying pathology in nonobliterating chronic postpneumonic pyogenic empyema, which permitted decortication and re-expansion in these cases, and more recently by Langston and Tuttle,[12] and Sampson and Burford[1] in their work on decortication for organizing hemothorax and posttraumatic empyema. The concept that the membrane which limited lung expansion was always due to inflammatory fibrosis of the pleura itself was proved erroneous in the case of tuberculous empyema by Delorme, and in the case of pyogenic empyema by Lilienthal, and was discussed by Sampson and Burford.[1]

In the light of this described pathology, the feasibility of decortication in some of these nonexpandable cases must be admitted, and has been proved in the cases presented herewith. Even in cases that have a favorable roentgenologic appearance, it is usually quite impossible to foretell preoperatively whether or not a satisfactory decortication can be accomplished. This calls for exploratory thoracotomy, which with modern techniques and skillful anesthesia does not usually entail any serious consequences or complications to the patient even in the presence of an open lesion on the opposite side. Preoperative thoracoscopy has been tried, but as one usually sees only a space lined with glistening hyalinized fibrin, and is unable to visualize the lung or lobar outlines, or in fact any distinguishing anatomic landmarks, we have not found it of very much value.

As for indications we are forced to depend so far on the theoretical deductions in which we assume that decortication and resultant re-expansion of the collapsed lung would be of benefit to the patient. Until our series of cases is larger we cannot define or limit the indications more clearly. We have set up tentatively the following list of possible indications:

1. To increase functional respiratory capacity when it is required to permit major collapsing procedure or resection for an open lesion on the contralateral side.
2. To avoid extensive thoracoplasty for the obliteration of the pleural dead space.
   (a) When pleural effusion is persistent or recurrent on attempted re-expansion. *(Collapsus imperatifs of Veran.)*
   (b) When a secondary pyogenic non-tuberculous empyema is present.
   (c) When attempted re-expansion is resulting in the shifting of the heart and mediastinum toward the chest wall, and creating overdistention dangerous to the opposite side.
3. Nonexpandability in the face of patient's desire to abandon pneumothorax, and avoid indefinite refills.

We have not included in our indications as yet tuberculous or tuberculous-pyogenic empyema, preferring for the present to observe our results in theoretically less hazardous cases. We have also so far avoided attempting decortication in any case which had a definite history of tuberculous empyema, or a pleural fluid positive for tubercle bacilli at any time during the course of pneumothorax treatments, even though presently dry or negative for these organisms.

The question arises as to the danger of subsequent reactivation of the old tuberculous focus or reopening of the cavity by the rapid re-expansion following decortication as compared with the more leisurely controlled re-expansion under fluoroscopic observation usually followed in readily re-expandable lungs on abandonment of pneumothorax. Only long-time observation on a large series of cases will determine the percentage expectancy of this eventuality. So far in our series we have attempted to obtain maximum re-expansion as soon

as possible postoperatively by means of an anterior upper, and posterior lower No. 45 French intrapleural tube connected with water-trapped drainage bottles. These tubes are usually removed in forty-eight to seventy-two hours. We have considered the possibility of maintaining a pneumothorax after decortication, and permitting a controlled re-expansion with a view to avoiding reactivation. However, it seemed that the ever-present danger of reopening old tuberculous foci on the pleural surface in the course of decortication demanded as rapid as possible obliteration of the pleural space for the prevention of a more serious complication, tuberculous empyema. So far we have no cause to regret this course of procedure, because none of our cases have developed a tuberculous empyema, or had persistent pleural fluid, although we have at least three times in the course of decortications opened into old tuberculous foci on the pleural surface. Neither have any of our cases to date had any reopening of the old cavities or reactivated lesions in the re-expanded lungs. We have noted under direct observation at the operating table after completion of decortication that the previous site of cavitation and maximum disease, due to parenchymal fibrosis and scarring of the visceral pleura in that area, does not re-expand well even with intratracheal positive pressure. The obliteration of the pleural space is accomplished by normal undiseased portions of the lung which push the fibrosed nonexpanding portion into the dome of the thorax or the costovertebral gutter. This has been further borne out by the postoperative roentgenograms which show the previous cavity-bearing area unexpanded.

In cases where decortication is indicated, and in which the duration of pneumothorax is not deemed sufficient to achieve healing of the original lesion, or where the roentgenograms indicate some danger of reactivating an old cavity, we have contemplated doing a partial decortication and re-expansion of the nondiseased portion of the lung, for instance an entire lower lobe, and leaving the diseased portion collapsed and encased in its limiting envelope. Then following maximum expansion of the nondiseased portion, a limited thoracoplasty would suffice to obliterate any residual pleural dead space. We have not yet encountered a suitable case for this procedure.

PREOPERATIVE PREPARATION

As the operation is elective and usually nonurgent, time and effort are not spared to bring the patient to operation in the best possible condition physically and physiologically, Attention is given to obtaining normal hemoglobin and red blood cell levels, normal blood protein, and augmented intake of vitamins. Bronchoscopy should be done on the patients to rule out active tracheobronchial tuberculosis, and cicatricial bronchostenosis. Vital capacity determinations should be made, and respiratory functional studies of both lungs by bronchospirometry are valuable and should be done if facilities are available. Thoracoscopy has been done on some patients, but as mentioned previously, does not seem to contribute a great deal of helpful information. Penicillin in dosage of 25,000 units every three hours is administered for forty-eight to seventy-two hours preoperatively and is continued postoperatively in increased dosage of 50,000 units every three hours until the vital signs have remained normal for forty-eight to seventy-two hours. Streptomycin has not been used routinely preoperatively in our cases to date. In Case 5 it was given for one week preoperatively in dosage of 1 Gm. per day, and continued for four weeks postoperatively at the same dosage level. In Case 4 it was given postoperatively in dosage of 1 Gm. per day for four

weeks. We are not prepared to comment on its efficacy, but will undoubtedly use it more freely in the future. We had planned to use it if cases showed any signs of fresh activation or tuberculous empyema following operation.

ANESTHESIA

We consider intratracheal anesthesia mandatory, and prefer ether-oxygen as the anesthetic of choice. At the end of operation the trachea is cleared by catheter suction through the intratracheal tube. If there is any indication that the bronchial tree is not perfectly dry, or that there are retained secretions or bronchial plugging, immediate bronchoscopic suction is done. This is repeated at any time during the immediate postoperative course, if atelectasis or retained bronchial secretions supervene.

OPERATION

It is unnecessary to give a detailed description of the operative technique as this has recently been so adequately done by Langston, Tuttle, and Crowley,[13] and by Sampson and Burford[1] in their descriptions of pulmonary decortication for organizing hemothorax. Our choice of approach has been through a posterolateral thoracotomy incision with resection of the fifth or sixth rib. The technique of the decortication is the same as described by these authors with this exception: in tuberculous cases when one approaches the diseased or former cavity-bearing area, one often finds that at this point the fibrous envelope is intimately adherent to the underlying lung, and at times shows evidence of tuberculous involvment. When this condition is encountered, the encasing plaque is separated from all directions toward the adherent area as far as easy separation is possible. The plaque is then cut around the adherent area leaving this adherent button attached to the underlying lung. In this way traumatizing the diseased portion of the lung and the danger of opening up old tuberculous foci are avoided. We should like also to emphasize the insistence of these authors on the complete freeing of the decorticated lung from all adhesions to the diaphragm and mediastinum, the freeing of the interlobar fissures, and the freeing of all adhesions on the visceral pleural surface which are causing enfolding or distortion of the lobe. In tuberculous lungs this freeing is, of course, carried out only over the more normal undiseased portions of the lung, and scarring over the diseased portion is not disturbed. At the end of decortication and freeing of adhesions the lung is gently and slowly re-expanded by intratracheal positive pressure to see that maximum expandability, especially of the more normal portions, is obtained. To maintain the expansion and drain postoperative effusion, two No. 45 French right angled latex suprapubic drainage tubes are placed into the pleural cavity through stab wounds in the third anterior and eighth posterior interspaces. If good expansion has been obtained and there is a minimum of residual pleural fluid as checked by x-ray these tubes are removed in forty-eight to seventy-two hours. During operation fluid is administered continuously through a cannulated leg vein using 5 per cent glucose in saline, and whole blood as indicated. Sufficient whole blood is given during and immediately following operation to replace all blood loss and restore a normal hemoglobin and hematocrit reading. Penicillin, 100,000 units in 20 c.c. of solution, is placed in the pleural cavity before closure of the operative wound.

POSTOPERATIVE CARE

Oxygen is administered for forty-eight to seventy-two hours postoperatively by oxygen tent or intranasal catheter. If the vital signs are normal and there are no contraindications, patients are permitted out of bed following removal of the expansion tubes from the pleural cavity. Deep breathing exercises are also started on this day, unless contraindicated by pathology in the opposite lung. Fluids by mouth are started postnausea, and regular diet resumed as tolerated.

CASE REPORTS

CASE 1.—M. S., a 30-year-old white woman, a nurse, was admitted to St. Vincent's Hospital Jan. 30, 1946, with temperature of 103° F. and complaint of pain in the left side of

the chest, dyspnea, and nausea. X-ray examination revealed left hydropneumothorax with fluid to the level of the sixth posterior rib. No aerated lung was visible on the left side. Right lung showed no pathology.

Past History.—The patient had entered the hospital in August, 1938, with the complaint of unproductive cough of five weeks' duration, loss of weight, and fatigue. X-ray views revealed a tuberculosis infiltration of the left apical region with a 2 cm. cavity, and sputum was positive for tuberculosis. She was transferred to a sanatorium where artificial pneumothorax was induced and a closed intrapleural pneumonolysis for apical adhesions was done. An excellent collapse was obtained, and sputum conversion followed shortly. She was discharged from the sanatorium in the summer of 1939 to continue pneumothorax treatment at home as an ambulatory case. She resumed work as a nurse about January, 1940, and continued to work steadily until the fall of 1945. During this time pneumothorax refills were given at intervals of ten days to two weeks. Sputum remained consistently negative and there were no clinical or roentgenologic evidences of activity. During the summer of 1945 decision was made to abandon pneumothorax and refills were stopped. The collapsed lung proved to be nonexpandable and pleural effusion developed with symptoms of pain and tension which required thoracenteses repeated at intervals of seven to ten days for relief. She had been having these thoracenteses up to the time of admission to the hospital in January, 1946.

Treatment.—Thoracentesis was productive of 750 c.c. of thin, amber, slightly cloudy nonodorous fluid from which was cultured *Staphylococcus albus.* It was negative for tubercle organisms on smear and culture. Repeated thoracenteses were done at two-day intervals, removing from 250 to 500 c.c. of fluid of the same character, and replacing in the pleural cavity at the completion of each tap 100,000 units of penicillin. After the fourth thoracentesis the vital signs returned to normal and symptoms subsided. Smears and cultures from subsequent taps were all negative for pyogens and tubercle bacilli. Despite the fact that pleural fluid reaccumulated, symptoms were minimal, and no further thoracenteses were done. The patient was discharged from the hospital, and remained under observation at home. She re-entered the hospital in June, 1946, for attempted decortication and reexpansion and operation was done on June 6, 1946.

Operative Note.—Left posterolateral thoracotomy incision with resection of the fifth rib was done. Fibrosed parietal pleura about ¼ inch thick was incised and the hydropneumothorax space entered. This space extended from the apex to the diaphragm and occupied about 50 to 60 per cent of the left pleural cavity. About 300 c.c. of slightly cloudy serous fluid were removed by suction, and several handfuls of gelatinous fibrin were removed from the bottom of the cavity and wiped from its lining walls. The cavity was lined by a firm, tough, well-organized fibrous plaque about ¼ inch in thickness with a smooth yellowish-white surface. The mid-portion of this plaque covering the collapsed lung was carefully incised until the visceral pleura was reached. The edges of the incised plaque were grasped with Allis forceps and elevated and it was found that a distinct line of cleavage could be developed between the inner surface of the plaque and the intact visceral pleura. By careful and painstaking blunt dissection with gauze tips, following this line of cleavage, it was possible to dissect this plaque off the entire surface of the collapsed upper and lower lobes. It was necessary to make a second intercostal thoracotomy incision in the eighth interspace to complete the dissection of the fibrous plaque from the lower portion of the lower lobe and the diaphragm. The plaque was then resected around its periphery at its angle of reflection onto the parietal pleura of the chest wall. The portion of the plaque on the parietal pleura was left in place. The lung lobes were then entirely freed up by separating the loose fine areolar adhesions between them and the remaining portions of the chest wall, mediastinum, and diaphragm, and the interlobar fissure was likewise separated. The lung was then expanded by intratracheal positive pressure. The lower lobe and the lower two-thirds of the upper lobe expanded fully and well. The upper one-third of the upper lobe, which was the seat of the original tuberculous lesion, was firm and fibrotic, and did not aerate. No. 45

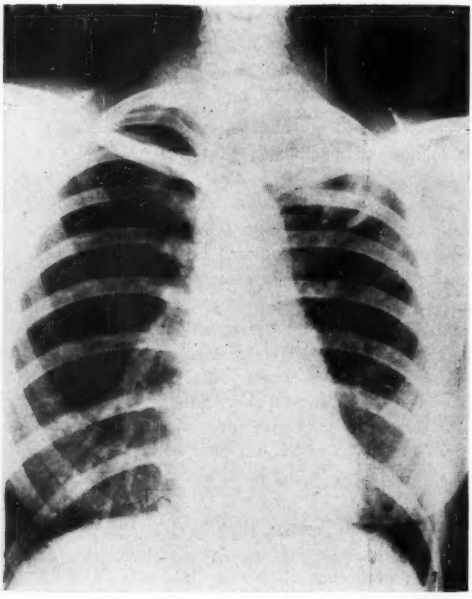

Fig. 1.

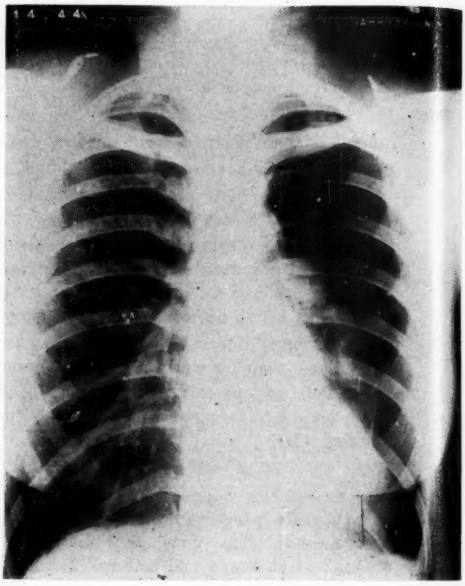

Fig. 2.

Fig. 1 (Case 1).—Original tuberculous lesion, 1938. Caseous-pneumonic process with cavitation in left upper lobe.

Fig. 2 (Case 1).—Status during pneumonthorax treatment, 1939 to 1945. Well-collapsed lung with fibrosis of upper lobe lesion. No pleural fluid.

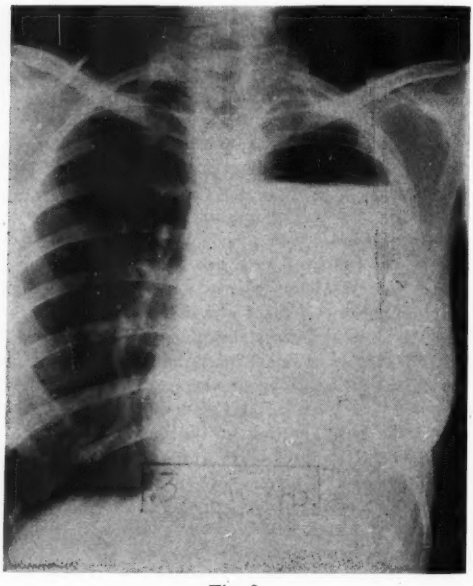

Fig. 3.

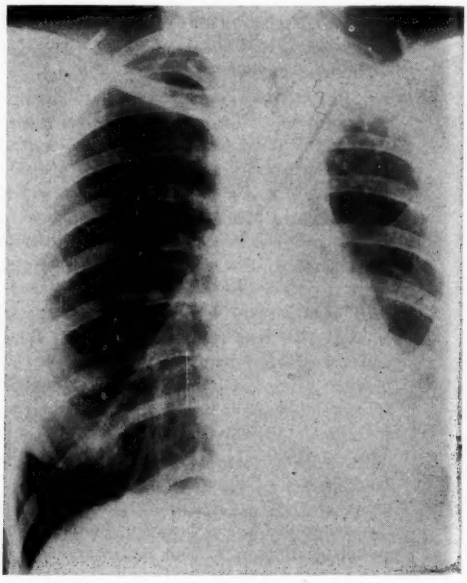

Fig. 4.

Fig. 3 (Case 1).—March 26, 1946, hydropneumothorax. Nonexpandable collapsed lung not visible. Before left decortication.

Fig. 4 (Case 1).—April 1, 1947, ten months after decortication and re-expansion of left lung. Pneumothorax space completely obliterated. Lung well expanded. Note supra-apical organized fibrous cap and unexpanded fibrotic lung at site of original lesion.

French latex right-angle drainage tubes were placed into the pleural cavity through stab wounds in the third anterior and seventh posterior interspaces. These were later connected with water-seal drainage bottles. The two thoracotomy wounds were closed using interrupted, layer sutures of chromic catgut, with continous fine silk for the skin closure. The lung was kept expanded by intratracheal, positive pressure until closure was completed. Continuous blood transfusion had been given during operation. Blood loss was minimal, and patient's condition at the close of operation was excellent.

Fig. 5 (Case 1).—Juxtapleural surface of peel removed at operation.

Postoperative Course.—Postoperative convalescence was most satisfactory. She was out of the oxygen tent on the third postoperative day and out of bed on the fifth postoperative day. Water-seal expansion tubes were removed on the fourth postoperative day. She continued to have a slight elevation of temperature to 100° F. until the thirteenth postoperative day, after which the vital signs remained normal. She was discharged on the sixteenth postoperative day ambulatory and in good condition. Discharge x-ray views showed an 85 to 90 per cent re-expansion of the left lung with a small residual area of hydropneumothorax, leaving eventually a small dense shadow at the left apex.

In October, 1946, she resumed part-time work as a nurse for a trial period of one month, following which she resumed full-time work which she has continued up to the present. At no time has there been any clinical or roentgenologic evidence of re-opening of the left apical cavity or reactivation of the tuberculosis. There has been no cough or sputum, and gastric washings have been consistently negative up to October, 1947.

Comment.—The indication for decortication in this case was obliteration of the pleural dead space resulting from a postpneumothorax nonexpandable lung and its accompanying recurrent effusions, once infected with pyogens, and the avoidance of multiple stage deforming thoracoplasty to accomplish this. The result to date has been most satisfactory.

CASE 2.—K. D., a 29-year-old white woman, was readmitted to Triboro Hospital in September, 1945, for treatment of a tuberculous cavity in right upper lobe, and recurrence of positive sputum.

Past History.—Original admission to Triboro Hospital in June, 1944, with history of onset with cough, sputum, and fever was in April, 1944. At that time the patient was in the third month of pregnancy. Sputum was positive for tuberculosis. X-ray examination showed a large thin-walled cavity in the left apex. Left pneumothorax was initiated in July, 1944, and closed intrapleural pneumonolysis was performed in September, 1944. During December, 1944, she was delivered of a full-term normal baby. February, 1945, a repeat pnuemonolysis resulted in complete collapse of the left upper lobe, cavity closure, and sputum conversion. She was discharged from the hospital in April, 1945, and continued pneumothorax treatments in the outpatient department. In July, 1945, sputum again turned positive and x-ray examination revealed a new thin-walled cavity in the right upper lobe. She was readmitted to Triboro Hospital in September, 1945.

Status on Admission.—General condition of the patient was good. Sputum concentrates were positive for tuberculosis. Sedimentation rate was 38 mm., vital capacity 1,600 c.c. Tomographic x-ray views of both lungs showed a 3 by 2 cm. cavity in the right apex, and no residual cavity in the collapsed left upper lobe.

Attempted therapeutic pneumothorax on the right was unsuccessful. Due to low vital capacity it was considered essential to obtain some re-expansion on the left before right upper thoracoplasty could be undertaken. Left pneumothorax was abandoned but after several months the left lung failed to re-expand appreciably. X-ray appearance suggested that a thick fibrous plaque covered the collapsed and enfolded lung. This was confirmed by thoracoscopy. Bronchoscopy in February, 1946, showed no intrabronchial pathology. In the meantime on strict bed rest general condition of the patient and x-ray appearance of the right lung cavity remained unchanged. Vital capacity had dropped to 1,500 c.c. Decision was made to attempt decortication and re-expansion of the left lung prior to right thoracoplasty. On April 16, 1946, under intratracheal ether-O_2 anesthesia a left thoracotomy and decortication of the left lung were performed.

Operative Note.—Left posterolateral thoracotomy incision was done with resection of the fifth rib and entrance to the pleural cavity through its periosteal bed. The lung was found about 60 per cent collapsed and broadly adherent to the cupola as well as to the posterior and anterior chest walls. The entire lung was encased in a thick grayish-white layer of hyalinized fibrous tissue which could be separated from the visceral pleura by sharp and blunt dissection, leaving an intact and normal-appearing visceral pleura. Over a small area across the upper lobe this peel was firmly adherent and on separation of its lower edge the pleura was torn and a small caseous area denuded. The freed edge of the peel was cut around this adherent arc leaving a round plaque about the size of a quarter attached to the lung. The lower edge of this plaque was sutured to the surrounding lung tissue to cover the denuded caseous area. Very little oozing was encountered, and the lung inflated readily except for the upper portion of the upper lobe which showed some puckering, and failure of expansion with positive intratracheal pressure in an area corresponding to the previously diseased, and at present fibrotic, parenchymal portion. The freely expanding part of the lower lobe and the lingula seemed to fill the chest completely. The pleura was then closed with interrupted sutures, after two L-shaped drainage tubes were placed into the pleural cavity, one in the second anterior interspace, and connected with water-seal bottles. The wound was closed in layers; 500 c.c. of whole blood were given during operation. The patient left the operating room in satisfactory condition.

Postoperative Course.—Postoperative x-ray views showed immediate and almost complete re-expansion of the left lung. The previous cavity area in the upper lobe did not re-expand, and in the ensuing months showed no sign of reopening or reactivation. The pleural space was obliterated except for a very small supra-apical fluid collection, which eventually partially absorbed and partially organized. The expansion tubes were removed at

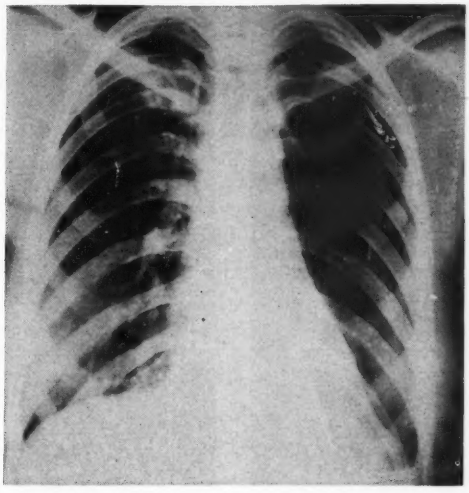

Fig. 6 (Case 2).—Jan. 4, 1946, left pneumothorax with nonexpandable lung. Right upper lobe cavity. Prior to left decortication.

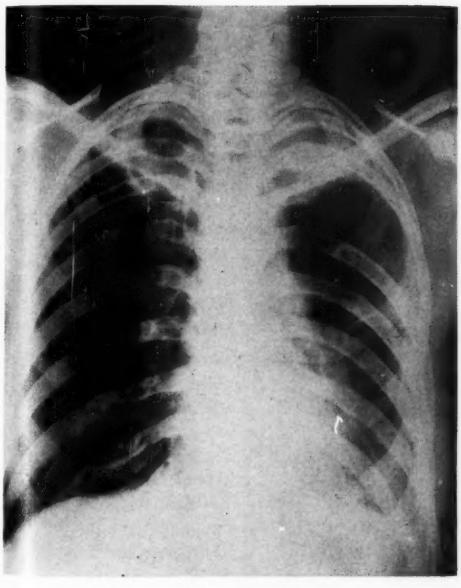

Fig. 7.

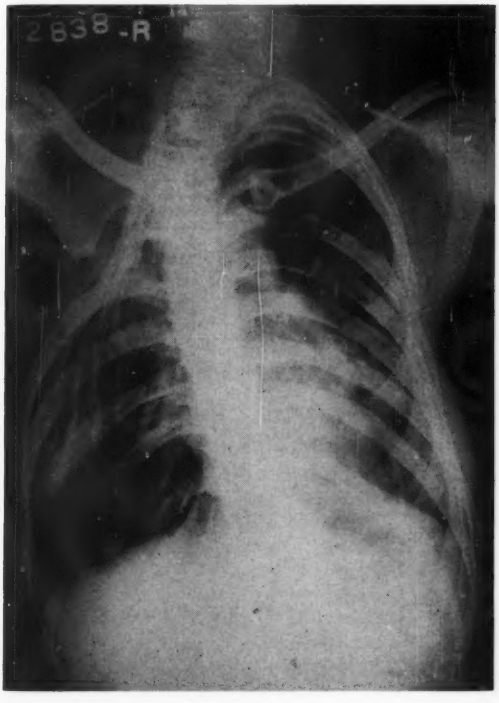

Fig. 8.

Fig. 7 (Case 2).—May [illegible] 1946, postdecortication. Complete expansion of left lung and obliteration of pneumothorax space. Note poor aeration of fibrosed left apical area, and increased size of right upper lobe cavity.

Fig. 8 (Case 2).—Sept. 3, 1947, seventeen months after left decortication. Fourteen months after right thoracoplasty.

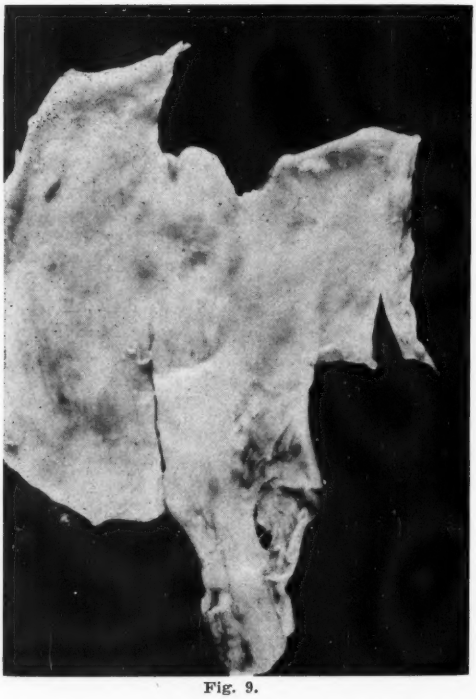

Fig. 9.

Fig. 10.

Fig. 9 (Case 2).—Fibrous plaque removed at decortication. Pearly hyalinized surface toward pneumothorax space. Deep notch at top marks cut around adherent area left attached to left upper lobe.

Fig. 10 (Case 2).—Fibrous plaque removed by decortication. Juxtapleural surface showing loose vascularized areolar layer.

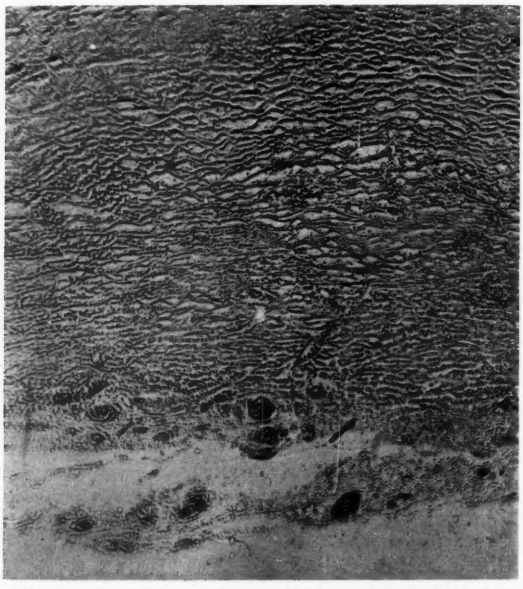

Fig. 11 (Case 2).—Microscopic section of plaque, showing thick hyalinized fibrin layer, and the juxtapleural loose vascularized areolar layer.

forty-eight and seventy-two hours, respectively, after operation. The clinical course was benign and there was no adverse effect on the contralateral lesion. About one month after decortication the vital capacity had increased slightly to 1,600 c.c., and by July, 1946, it had increased to 1,900 c.c.

On July 16, 1946, an upper stage right thoracoplasty with removal of the upper three ribs was performed. This was followed on July 31 by a second stage in which the fourth, fifth, and sixth ribs and the lower one-third of the scapula were removed. Sputum decreased but remained positive and a small residual cavity was visualized. On Dec. 3, 1946, a revision of the thoracoplasty was done. Following this operation cough and sputum practically disappeared, but three-day concentrates and gastric washings continued to show tubercle bacilli, and tomograph studies demonstrated a small residual cavity in the costovertebral gutter. General condition was excellent, and there was no dyspnea. The patient had gained twelve pounds since the original operation.

On May 2, 1947, a second revision with intrapleural apicolysis and supra-apical muscle transplant finally achieved cavity closure and sputum conversion.

Comment.—The object of decortication and re-expansion in this case was to achieve sufficient increase in functional capacity to permit a permanent collapsing operation urgently indicated for cavity closure on the contralateral side. That the procedure was eminently successful has been attested by the subsequent course of events.

CASE 3.—O. S., a white man aged 33 years, was admitted to Triboro Hospital Feb. 17, 1947, with right pneumothorax of fourteen years' duration and nonexpandable lung.

Past History.—From previous history it is noteworthy that the disease started in 1933 when the patient developed a cavitary tuberculosis of the right upper lobe for which he was hospitalized and artificial pneumothorax instituted. A pneumonolysis was performed in 1939 and the positive sputum converted. In 1935 he was discharged from the hospital with the pneumothorax maintained. He was well until 1939 when he developed a disseminated lesion in the left upper lobe, for which he was treated on conservative therapy at Bellevue Hospital and at Sea View Hospitals. According to the patient's statement, a cavity on the left side developed and disappeared. In 1942, the cavity on the left side reopened and the sputum turned positive. Re-expansion of the right pneumothorax was attempted unsuccessfully, resulting in a temporary hydropneumothorax. For this last episode the patient was hospitalized at Triboro Hospital from November, 1942, to April 1943, when he was discharged with negative sputum. The patient was well for three years. The pneumothorax was maintained during this time with monthly refills to avoid symptoms of tension and recurrence of hydrothorax.

He was admitted to Triboro Hospital March, 1946, and right phrenemphraxis was done to help obliterate right pneumothorax space. He was discharged in June, 1946.

Status on Admission.—Sputum at the time of admission was still negative and all laboratory findings were normal. Vital capacity was 3,000 c.c. Bronchoscopy March 1, 1947, showed a collapsed slitlike right main bronchus but no stenosis. Reviewing the old x-ray pictures it was noticed that there was some re-expansion of the lung between May, 1945, and March 17, 1947. The x-ray view taken on May 18, 1947, showed an almost completely collapsed lung dotted with calcified foci. This view showed also an anterior herniation of the left lung into the right pneumothorax space. The x-ray picture of March 17, 1947, showed the right lung about 70 per cent expanded. The right lower lung field showed linear fibrosis and there was a calcification over the entire lung. The apex of the right lung showed emphysematous blebs and superimposed over the right lung field just beneath the clavicle one noted a semicircular line suggesting the herniation of the left lung through the anterior mediastinum into the right pneumothorax space. At the base of the pneumothorax space there was a low fluid level noted.

Fig. 12.

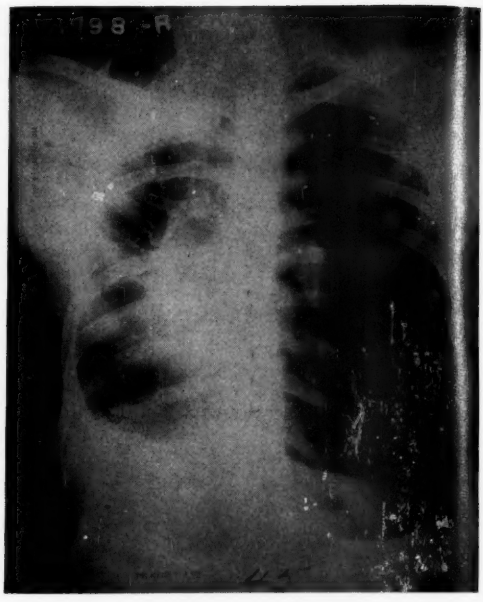

Fig. 13.

Fig. 12 (Case 3).—March 17, 1947, predecortication. Hydropneumothorax, right. Non-expandable lung after fourteen years of artificial pneumothorax.

Fig. 13 (Case 3).—Aug. 12, 1947, five months after decortication. Complete obliteration of the right pleural space.

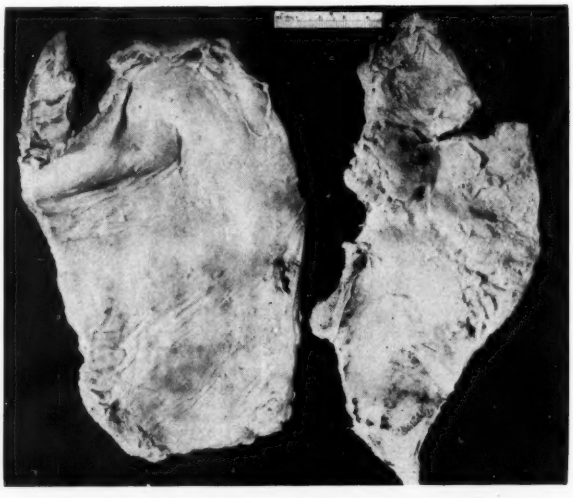

Fig. 14 (Case 3).—Fibrous plaque removed by decortication. Left is plaque removed from parietal pleura. Right is plaque removed from visceral pleura.

Operative Note.—The decortication was performed March 24, 1947, under intratracheal general oxygen-ether anesthesia. The operative procedure in this case was rather an extensive one.

A right posterolateral thoracotomy incision was made with resection of the sixth rib. The pleural cavity was entered through the periosteal bed of the sixth rib. The parietal pleura was about ¼ inch thick and composed of hyalinized fibrin. In the pleural cavity there were about 300 to 400 c.c. of clear, thin fluid slightly tinged with methylene blue, which had been injected previously. The hyalinized fibrin plaque covered the entire pleural space. The lung was moderately expandable under positive pressure. After removal of the fluid the fibrin plaque over the lower lobe was incised down to the visceral pleura and a plane of cleavage developed between the inside of the plaque and the surface of the lung. No real loose plane of cleavage could be found and dissection was tedious and difficult. The thick hyalinized plaque was stripped off the chest wall and the upper half dissected around the apex with extreme difficulty. In dissecting the plaque away from the upper mediastinum most of the mediastinal pleura came with it, exposing the venacava and the phrenic nerve. After this portion of the plaque had been freed from the chest wall and mediastinum it was dissected off the remainder of the upper lobe. A small portion of the posterior apical area was composed of a large emphysematous bleb and this came away attached to the plaque. The raw surface of the lung thus exposed did not bleed and it was repleuralized by inverting the edges of the visceral pleura with a fine catgut suture. A similar dissection was then carried out to strip the plaque from the lower half of the chest wall and the lower and middle lobes. Here again the dissection was extremely difficult, especially along the mediastinal surface and over the diaphragm, and finally, a patch of the plaque about 3 inches in diameter was left attached to the diaphragm. It was necessary also to leave a similar patch about 2 inches in diameter on the lateral surface of the lower lobe. The lung was then re-examined by positive intratracheal pressure. The remaining portion of the upper lobe aerated and expanded very poorly; the lower lobe expanded fairly well and filled about one-half of the chest cavity. The pleural cavity and lung were then sprinkled with streptomycin-plasma mixture and 100,000 units of penicillin were placed into the pleural cavity. Two No. 45 right angle latex drainage tubes were introduced into the pleural cavity in the third anterior interspace, and in the ninth lateral interspace, to be later connected with water-seal bottles. The thoracotomy wound was then closed in layers with interrupted catgut sutures.

Note.—The procedure was extremely rough and shocking but the continuous blood transfusion throughout the operation maintained the patient's pressure at a level about 100 and at the close of the operation the pressure was 110/70. He returned to the ward in good condition.

Postoperative Course.—Postoperatively the patient received daily 1 Gm. streptomycin and 150,000 units penicillin parenterally, and 50,000 units penicillin and 400 mg. streptomycin locally into the pleural space for one week. The lung did not show immediate complete re-expansion. There was residual pneumothorax at the apex of the right pleural cavity, and also evidence of air leakage from the lung noticed in the water-seal system for the first four postoperative days, undoubtedly due to the injuring and suturing of emphysematous lung tissue during the operative procedure. Fluid drainage was minimal. The lower drainage tube was removed on the fourth postoperative day. The upper tube was removed on the eighth postoperative day, forty-eight hours after evidence of gross air leakage ceased. The apical residual pneumothorax was deflated at twenty-four to forty-eight hour intervals by removal of 30 to 50 c.c. of air to maintain satisfactory negative pressures. On one of these thoracenteses the herniated left lung was punctured by the needle and the left traumatic pneumothorax resulted. This tension pneumothorax was treated by continuous deflation for forty-eight hours. The left lung re-expanded and the episode passed without any sequelae. The right pleural space subsequently became entirely obliterated (see postoperative x-ray picture, Fig. 13, Aug. 12, 1947). The re-expanded right lung filled about four-fifths

of the space. The anterior upper portion was occupied by a herniated portion of the left lung, posterior to which there was an apical cap of thickened parietal pleural and organized fibrin. The costophrenic angle was obliterated, and the shift of the heart and mediastinum to the right was about the same as it was preoperatively.

Comment.—The objective of obliteration of the right pleural space after artificial pneumothorax of fourteen years was achieved. The result was highly satisfactory from the patient's standpoint, as he was completely relieved of symptoms of tension and dyspnea, and stated that breathing was easier. He was in addition relieved of the necessity of periodic refills and thoracenteses for recurrent fluid. He has been discharged from the hospital for the past four months and is able to work at his profession as a commercial artist.

CASE 4.—V. J., a 34-year-old white woman, was admitted to Triboro Hospital Dec. 16, 1946, from the outpatient department with the complaint of occasional intractable coughing spells.

Past History.—Onset of tuberculosis was in the spring, 1939. Artificial pneumothorax was induced in April, 1939, for cavitary lesion of the left upper lobe. Pneumonolysis was done in November, 1939. Adequate collapse and sputum conversion were achieved in December, 1939. During the intervening years until this admission she was in and out of sanatoriums and hospitals, and was followed a greater part of the time in the outpatient department of Triboro Hospital. Pneumothorax was maintained and sputum was always negative. During the year previous to admission attempts to abandon pneumothorax and re-expand the lung were unsuccessful due to symptoms of tension, pulling, intractable cough, and development of small intrapleural effusion.

Present Admission.—Pulse and temperature were normal. Blood count and urinalysis were also normal. Sedimentation rate was 3 mm. per 1 hr. Sputum and gastric washings were negative for tubercle bacilli on smear and culture. X-ray examination showed an 80 per cent left pneumothorax with a small pleural effusion. Tomographic plates of both lungs revealed no cavitary pathology. Bronchoscopy revealed no intrabronchial cicatricial stenosis, but the left main bronchus was collapsed to a slitlike opening. Thoracoscopy done in February, 1947, showed the pneumothorax space to be lined with hyalinized fibrous tissue which obliterated all fissures and other anatomic landmarks. Vital capacity was 1,700 c.c. The patient insisted firmly on abandoning pneumothorax refills after 7½ years. Decortication was suggested and accepted by the patient with thoracoplasty to follow if re-expansion was unsuccessful. Decortication was performed on Feb. 26, 1947.

Operative Note.—Operation was performed with intratracheal general oxygen ether anesthesia. A left postero-lateral thoracotomy incision was made. The sixth rib was resected from the transverse process to the anterior axillary line. Incision was made into the pneumothorax space through the periosteal bed of the resected rib. The parietal pleura was thickened to about ¼ inch, and cut with a hard, gritty feel. When the pneumothorax space was entered it was found to be completely dry and lined with a pearly white, thick fibrous plaque. The lung was wholly collapsed against the mediastinum and posterior costovertebral gutter, and held down by this thick fibrous plaque. There was no expansion of the lung whatever, even under positive intratracheal pressure. The mid-portion of the plaque over the lung was carefully incised until the visceral pleura was reached. Between the inside of the fibrous plaque and the visceral pleura a line of cleavage was developed in which, by careful blunt dissection with gauze and with a gloved finger, the plaque could be separated from the visceral pleura. The plaque was divided thus, away from the lung and mediastinum anteriorly as far as its reflection on the anterior chest wall and posteriorly as far as its reflection on the posterior chest wall and upward along these lines to the extreme apex. The plaque was then cut along its angle of reflection to the chest wall and removed. Corresponding dissection was carried out on the lower half of the

plaque dissecting it free from the lower lobe and the diaphragm, and then removing it. Attempt was then made to re-expand the lung by positive intratracheal pressure, but it did not re-expand very well. There was a rather marked intrapleural fibrosis across the medial aspect of the midlung field having a stellate character and broadening out into a fibrotic band about 1 inch in width which ran posteriorly. This seemed to keep the lung from expanding in this area, and it was impossible to free it without opening into the lung tissue. Several small bands of adhesions on the lower lobe and at its medial surface were dissected free and this portion of the lung re-expanded fairly well. Also the anterior and upper parts of the upper lobe expanded fairly well. At one point, the dissection of the fibrous plaque from the lung loosened a small calcified tuberculous nodule about 2 mm. in diameter. This was removed. There was no purulent or caseous reaction around it. The re-expansion of the lung was disappointing. I would estimate that about only a 50 per cent re-expansion of the lung was achieved. No attempt was made to remove the fibrous lining from the parietal pleura. Two No. 45 French right angle perforated drainage tubes were placed into the pleural cavity through stab wounds in the third anterior and eighth posterior interspaces, to be later connected with water-seal bottles. The thoracotomy wound was closed in layers using interrupted No. 1 chromic catgut sutures throughout. During operation 1,000 c.c. of whole blood, and 500 c.c. of 5 per cent glucose in saline were administered. The patient left the operating room in good condition.

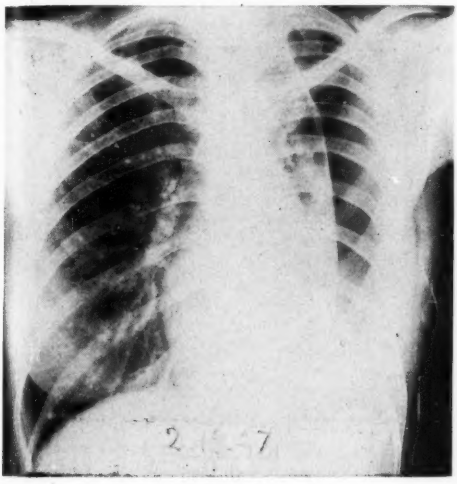

Fig. 15.

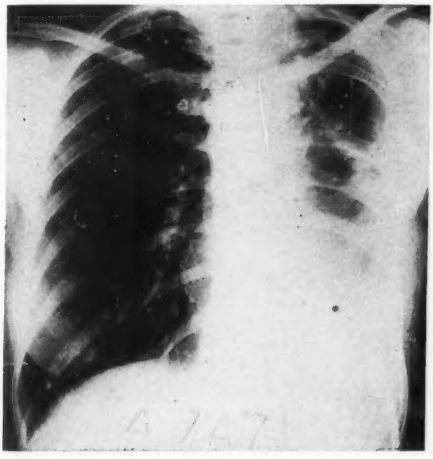

Fig. 16.

Fig. 15 (Case 4).—Feb. 18, 1947, nonexpandable lung after seven years of artificial pneumothorax.

Fig. 16 (Case 4).—April 7, 1947, six weeks after decortication. Complete re-expansion of left lung with obliteration of pleural space.

The postoperative course was uneventful. The lung re-expanded readily and filled the hemithorax. This was surprising and gratifying in view of the less optimistic expectancy during operation. The drainage tubes were removed forty-eight and seventy-two hours, respectively, after operation. The vital capacity did not increase in the first four post-operative weeks over the original 1,700 c.c. but the patient stated that breathing was much easier and felt more efficient. She was discharged May 2, 1947, from the hospital. She gained seven pounds after operation over her original weight on admission. Up to October, 1947, there has been no evidence of reactivation or reopening of old cavities.

Comment.—This case represents a classical indication for decortication of a nonexpandable lung. The tuberculous process seemed to have been arrested for over seven years. No bronchial pathology or extensive parenchymal fibrosis could be held responsible for the nonexpansion. The cause of the nonexpandability seemed to be chiefly the constricting fibrous coating of the pleura due to the chronic pneumothorax. The annoying symptoms and the intense desire of the patient to abandon pneumothorax refills dictated some surgical procedure for the obliteration of the pleural space. The successful surgical re-expansion spared the patient an extensive multiple stage thoracoplasty with its resultant deformity. No adverse effect has occurred up to eight months postoperatively on the course of the arrested tuberculosis.

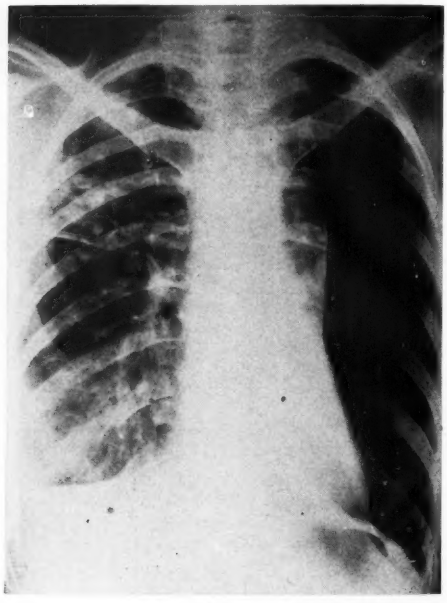

Fig. 17.

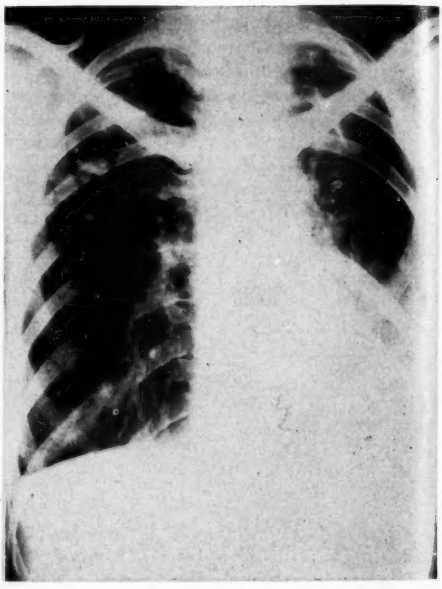

Fig. 18.

Fig. 17 (Case 5).—March 29, 1947, predecortication. Completely collapsed nonexpandable lung after four years of artificial pneumothorax.

Fig. 18 (Case 5).—Aug. 27, 1947, 2½ months after technically complete decortication. Residual pneumothorax space and failure of re-expansion of left lung due to parenchymal fibrosis.

CASE 5.—M. K., a white 33-year-old woman, was re-admitted to Triboro Hospital, March, 1946, because of fresh spread of tuberculous lesion in right upper lobe.

Past History.—Original admission to Triboro Hospital was in June, 1942, with a history of onset of symptoms and diagnosis of pulmonary tuberculosis in September, 1941. The patient had had no treatment prior to this admission. X-ray pictures showed diffuse nodular infiltration of the right apex and left upper lobe; a like type of lesion plus small cavities in the left lower lobe. Sputum was positive for tuberculosis. Treatment of bed rest until March, 1943, when a left pneumothorax was induced. In early 1944 a fresh exudative lesion occurred in the right upper lobe. Bronchoscopy at this time was negative. The patient's lesion on the right showed resolution on strict bed rest. She was discharged to the

outpatient department in 1945 after three years of sanatorium treatment. Sputum was negative on concentrate, but persistently positive on culture. Left pneumothorax was maintained.

Present Status.—There was a fresh right upper lobe lesion with cavitation. Sputum was positive.

Treatment.—In April, 1946, marginal pneumothorax was instituted on the right. This had to be abandoned in September, 1946, because of tension pneumothorax. Refills on the left side were abandoned, but the lung failed to re-expand up to May, 1947. Vital capacity was 900 c.c. Bronchoscopy was negative. The patient was considered for a left decortication because of the low vital capacity, and desirability of permanent collapsing operation for right upper disease. It was felt preoperatively that the chances of good re-expansion with decortication were poor because of the diffuse character of the original lesion in the left lung and x-ray evidence of marked parenchymal fibrosis. Fig. 17 shows an x-ray view of the preoperative condition.

Operative Note.—Operation was done on June 9, 1947. The lung was found completely covered and bound down by a thin hyalinized fibrous plaque which was completely separated from the underlying visceral pleura with ease. The lung was completely decorticated, and freed from all adhesions to the diaphragm, pericardium, and mediastinum. The visceral pleura was thin, and in the process of decortication a few tuberculous foci immediately beneath the visceral pleura were denuded, but no caseous material escaped. It was immediately noted on completion of the freeing of the lung that there was marked parenchymal fibrosis. Intratracheal positive pressure failed to re-expand the lung or to aerate it to any satisfactory degree. After dusting the pleural surfaces with streptomycin-plasma mixture, and placing two intercostal expansion tubes, the thoracotomy wound was closed in the usual manner. Streptomycin was administered for seven days preoperatively in dosages of 1 Gm. per day, and continued for four weeks postoperatively at the same dosage level.

The postoperative course was completely benign and uneventful. The lung, however, showed only slight re-expansion, and the pneumothorax space was about as large as preoperatively (Fig. 18). There was no empyema or recurrent fluid formation and the operative wound and drainage tracts healed per primam.

Comment.—This case represents a failure to achieve lung re-expansion or obliteration of the pneumothorax space following complete decortication. The failure was due, we believe, to marked parenchymal fibrosis of the collapsed lung.

SUMMARY

Attention is called to the possibility of a new surgical approach to the problem of the postpneumothorax nonexpandable lung by means of decortication. The literature on older and more recent uses of pulmonary decortication for other indications is discussed. The pathologic factors responsible for some cases of nonexpandability, which provide the theoretical background for the use of the procedure in tuberculosis, are analyzed. Tentative indications for use of the procedure are outlined. Preoperative and postoperative care is delineated. For detailed description of operative technique reference is made to recent articles by Sampson and Burford,[1] and by Tuttle, Langston, and Crowley.[12, 13]

Detailed case histories of five patients who have had surgical decortication for postpneumothorax nonexpandable lungs are presented. One case was a failure in that obliteration of the pleural space was not obtained, and very little re-expansion of the lung resulted in spite of a complete technically successful decortication. This failure was due to marked parenchymal fibrosis of the lung. Four cases were completely successful in accomplishing the objective of the

procedure. In Cases 3 and 4, the objective was the abandonment of pneumothorax refills and the obliteration of the pleural space without resort to multiple stage thoracoplasty. In Case 1 the indication was obliteration of a postpneumothorax space subject to recurrent pleural effusions, which had once been infected with pyogens and sterilized by penicillin therapy. In Case 2 the objective was an increase in the functional capacity to permit urgently indicated thoracoplasty for contralateral cavity. The clinical results in these four successful cases have surpassed expectations. These four cases have had periods of postoperative observation of sixteen months, eighteen months, eight months, and seven months, respectively. One patient (Case 2) has subsequently undergone four stages of thoracoplasty, revision, and apicolysis to achieve cavity closure and sputum conversion for contralateral cavity. There have been no serious postoperative complications. There has been no tuberculous empyema or wound infection. There has been no clinical or roentgen evidence of reopening of cavities or reactivation of tuberculosis in the re-expanded lungs. Three patients have been out of the hospital for several months, enjoying an "apparently arrested" status, leading normal lives, and working. The fourth patient (Case 2) is ready for discharge from the hospital in an apparently arrested status.

CONCLUSIONS

Pulmonary decortication appears to be a useful procedure to add to the surgical armamentarium for dealing with the problem of the postpneumothorax nonexpandable lung.

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VAGUS NERVE SECTION IN THE TREATMENT OF PEPTIC ULCER

Abdominal Versus Thoracic Approach

Joseph A. Weinberg, M.D. (by invitation), George W. Campbell, M.D., and Robert B. Voelker, M.D. Van Nuys, Calif.

THE widespread interest in the operation of vagus nerve section devised by Dragstedt[1] for the treatment of peptic ulcer is as much an expression of dissatisfaction with other methods of surgical treatment as it is an enthusiasm for the new procedure. The expression of dissatisfaction with other methods is not surprising when it is considered that the best of the tried methods is one in which most of the stomach is removed to eradicate a relatively benign lesion. The enthusiasm for the new procedure, based upon the excellent immediate results, is being countered with a healthy opposition which asks that its acceptance await a period of some years in order to ascertain the permanency of relief. We are in complete accord with that admonition. It is obviously impossible to discuss permanency of results in the group of patients to be reported here, as the time since operation varies from twelve to twenty months. Sixty cases are included in this group, thirty by the abdominal and thirty by the thoracic approach. This division was arbitrarily made in order to compare the relative advantages and disadvantages of the two methods. It is not easy to evaluate the results in the disease of peptic ulcer, and the comparison of results becomes more difficult when the evaluations are made by different clinics. One may arrest hemorrhage, relieve obstruction, or relieve abdominal distress without effecting complete relief from symptoms referable to the psychic phase of this psychosomatic disease. Unfortunately, the same standards are not universally used in determining the degree of relief, and this must be considered in evaluating the results of this and other operations. Classifications of complete relief, partial relief, and failure have little meaning unless one states what is meant by these three types of results.

The operation of vagal denervation of the stomach is not a simple one. Denervation is not always attained because of the difficulty of working in the relatively unfamiliar region of the lower mediastinum and the variability of the pattern of the vagus nerves, which make it difficult to be certain that the nerves are adequately interrupted. Many of the objections which have been expressed against the operation should be charged to faulty performance rather than to failure of the procedure to achieve its aim when properly performed. It is this phase of the problem that is dealt with in this presentation.

VAGUS NERVE PATTERN

It is significant that the failures with both abdominal and thoracic approaches were limited to the early cases. This may be explained in part by a

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lack of familiarity with the variations in nerve patterns in our early experience. It has been observed in a study of more than 100 cadavers that the vagus nerves in the lower mediastinum fall into three main classes.

The most common type is that in which the left nerve is found to be a single main trunk in the lowest part of the esophagus, usually between the levels of 2 and 6 cm. above the hiatus. The right nerve is also a single trunk in a slightly lower area, between 2 and 4 cm. above the hiatus. A branch occasionally connects the right and left vagus nerves between the levels of 5 and 8 cm. above the hiatus. Seventy-four per cent fall into this group.

The next most common type is that in which the left vagus nerve fibers consist of a main nerve with a smaller nerve within 1 cm. of the main nerve but entirely separate from it from the level of 7 or 8 cm. above the hiatus down to the stomach. There may be small intercommunicating branches between the two. The right nerve in these cases does not differ from the common type and in general shows more branching than the left in the level between 5 and 10 cm. above the hiatus. Occasionally, it courses down the esophagus on to the stomach and celiac plexus as several branches. These branches are usually sufficiently close together to make it easy to pick up all branches at the same time when separating the nerve from the esophagus. The right nerve is loosely attached to the esophagus and is easily left behind when the esophagus is lifted from the mediastinal bed.

The third group, consisting of wide departures from the usual pattern, makes up 4 per cent of 100 specimens examined. In two of the specimens the left vagus nerve was partially submerged in the muscle. In the third specimen, the left vagus nerve was divided into five segments of about equal size. The fourth specimen showed a branch of the right vagus nerve which was intimately attached to areolar tissue of the mediastinum and free from the esophagus. This separate filament passed through the hiatus. We have seen no instances of vagus nerves passing from the thorax into the abdomen except through the esophageal hiatus.

One should not begin the separation of the vagus nerve from the esophagus too close to the esophageal hiatus. There are two reasons for this. First, one is likely to miss the fibers which leave the esophagus 1 or 2 cm. above the hiatus. Second, the left vagus nerve is intimately adherent to the esophagus in the level between 0 and 1 cm. above the hiatus and is not readily picked up in this area.

INDICATIONS FOR OPERATION

All of the patients in this series were diagnosed as having duodenal or stomal ulcer and were referred to surgery by the medical gastroenterology service because of hemorrhage, obstruction, intractable pain, or a combination of these indications. The ages of the patients ranged from 20 to 69 years.

PREOPERATIVE MANAGEMENT

Most of the patients who come to operation are in a poor state of nutrition because of dietary restrictions over a long period of time, obstruction, hemorrhage, or a combination of these factors. Protein deficiency is corrected by a

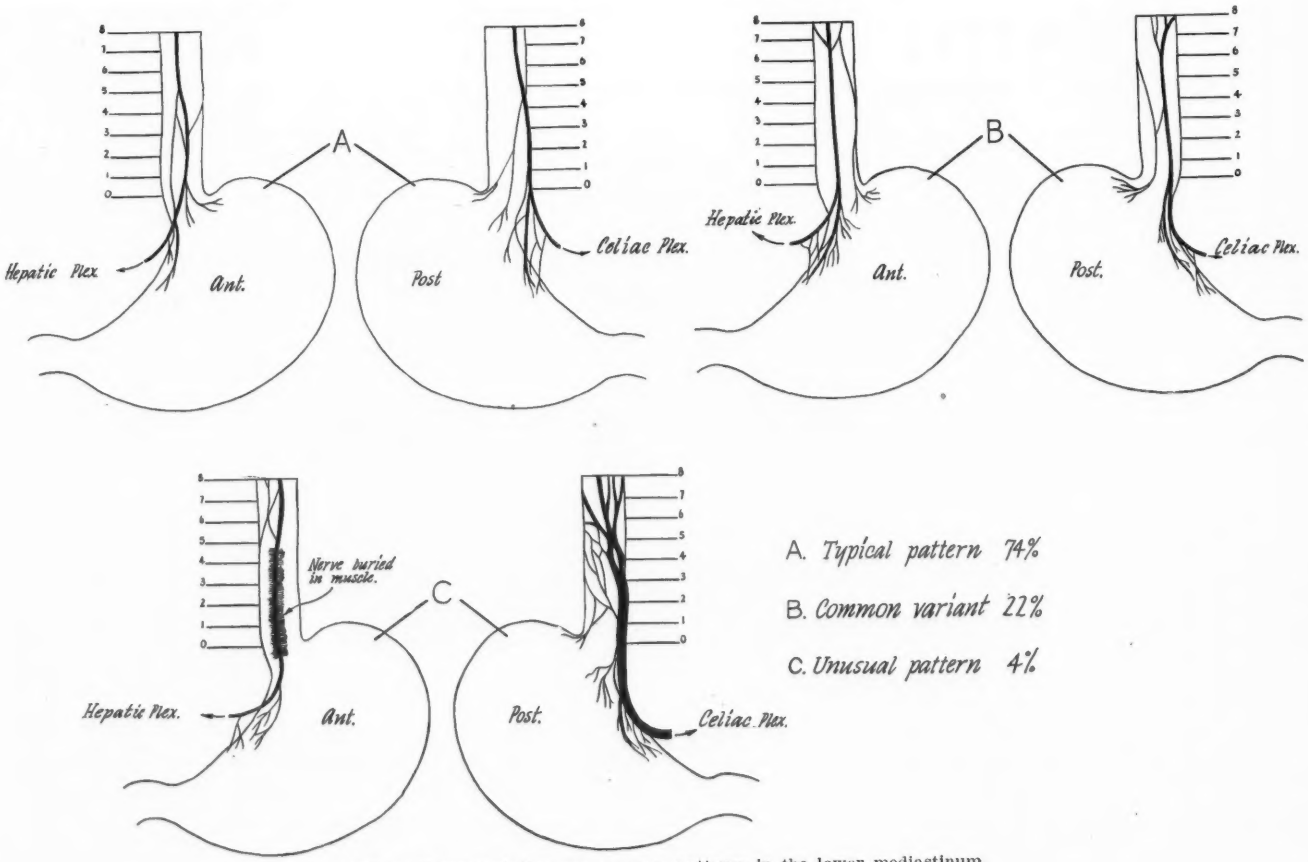

Fig. 1.—Representative vagus nerve patterns in the lower mediastinum.

high protein diet and amino acid infusions and blood deficiencies are corrected by transfusions. Vitamin C, 300 to 500 mg., is administered daily for at least seven days prior to surgery. The importance of correcting these deficiencies cannot be overemphasized. We are convinced that some of our early difficulties were the result of failure to appreciate the degree of malnutrition.

The insulin hypoglycemia test was used routinely before and after operation in our first cases but is now performed only postoperatively to determine the completeness of vagus nerve section. The test amounts to hypoglycemic shock and is distressing to the patient. Most of the patients subjected to the test have shown evidence of severe hypoglycemia, manifested by weakness, fainting, perspiration, hunger, and shaking. There is also the theoretical danger of inducing severe bleeding in the preoperative ulcer patient, although we have not yet seen what could be termed dangerous bleeding. For these reasons it is questionable whether the test should be used except in those cases in which failure to accomplish denervation is suspected.

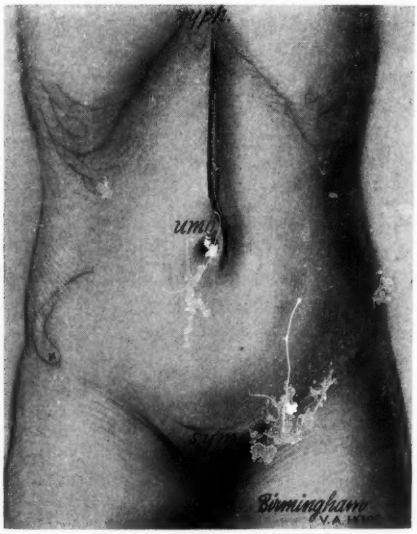

Fig. 2.—Skin incision; the abdominal incision.

The stomach and bowel are emptied preoperatively by giving a liquid diet beginning twenty-four hours before surgery, a saline cathartic in the morning, and a normal saline enema in the evening of the day before surgery. A Levine tube is introduced into the stomach prior to operation. All patients are given penicillin intramuscularly from three days before operation to five days after operation.

TECHNIQUE OF ABDOMINAL APPROACH

Spinal anesthesia is used because of its action in promoting peristalsis and in facilitating exposure.

An incision is made through the abdominal wall 2 cm. to the left of the midline, extending from the ensiform cartilage to 2 cm. below the umbilicus. The stomach, duodenum, and other upper abdominal viscera are examined, noting the degree of activity of the ulcer. It is impossible to determine the degree of pyloric obstruction by inspection or palpation and roentgenologic examination must be relied upon for this information.

The left lobe of the liver is retracted upward and to the right by means of a broad retractor designed for this purpose. Adequate exposure of the hiatus may be obtained without division of the left hepatic ligament. The

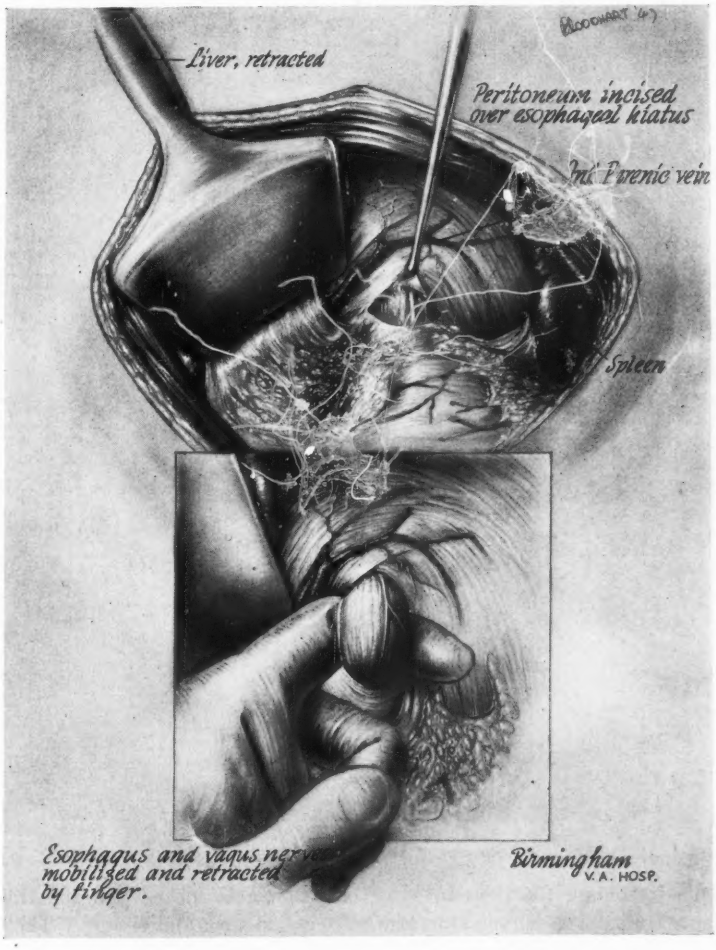
Liver, retracted
Peritoneum incised over esophageal hiatus
Int. Phrenic vein
Spleen
Esophagus and vagus nerve mobilized and retracted by finger.
Birmingham V. A. HOSP.

Fig. 3.—Left lobe of liver retracted cephalad and to right. Peritoneum over esophageal hiatus incised, exposing esophagus. Insert shows esophagus retracted by index finger.

stomach and intestines are retracted with the gloved hand and a transverse incision 2 to 3 cm. in length is made through the peritoneum covering the esophageal hiatus. The left subphrenic vein, which courses transversely just anterior to the hiatal ring, is carefully avoided. The right index finger

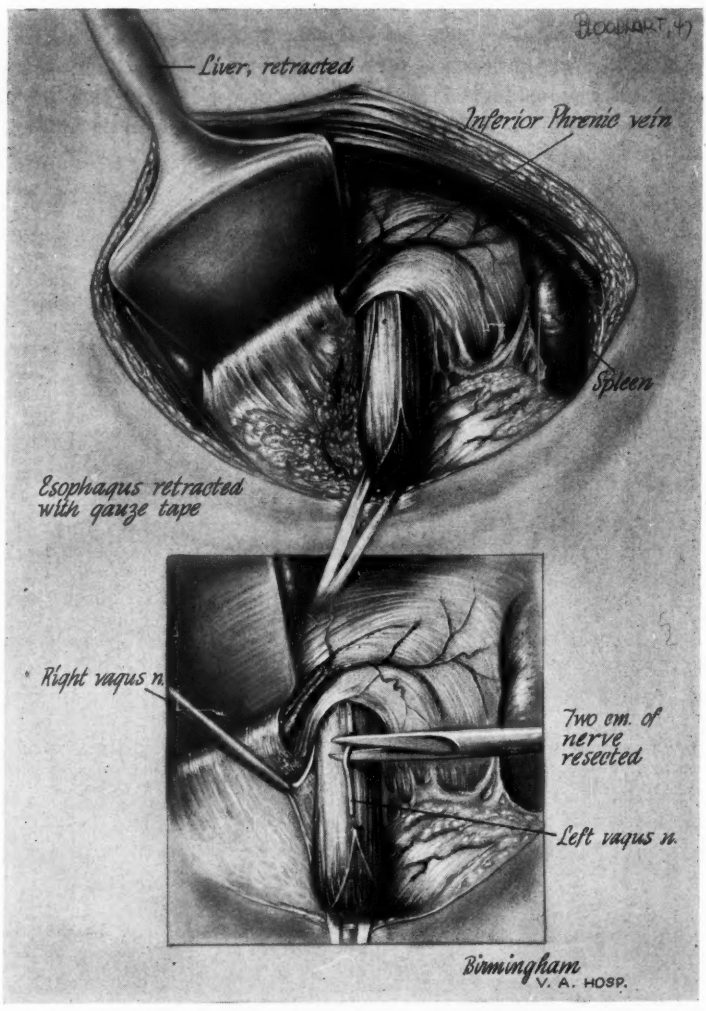

Fig. 4.—Resection of nerve segment between ligatures.

is inserted through the incision into the esophageal hiatus and separates the esophagus and vagus nerves from the adjacent mediastinal tissues. The esophagus, with the attached nerves, is then drawn into the abdomen by means of the flexed index finger, and an empty Penrose rubber tube is placed around

it for traction. At least 6 cm. of the esophagus can be brought into view by this maneuver. The nerves are identified by palpation and inspection. Clamps are placed 2 cm. apart on each of isolated vagus nerves and the segment between the clamps is resected. The divided ends of the nerve are ligated with cotton suture. Small nerve filaments are transected without ligation. The resection of a segment is usually performed between 2 and 4 cm. proximal to the hiatus, since the nerves are more integrated at this level. After division of the vagus nerves has been completed, the penrose tube is withdrawn and the esophagus is returned to its bed. The peritoneal incision over the hiatus is not sutured. Gastroenterostomy is performed if there is existing or anticipated obstruction at the pyloric ring. The abdominal wound is closed in layers, using interrupted sutures for the fascial layers.

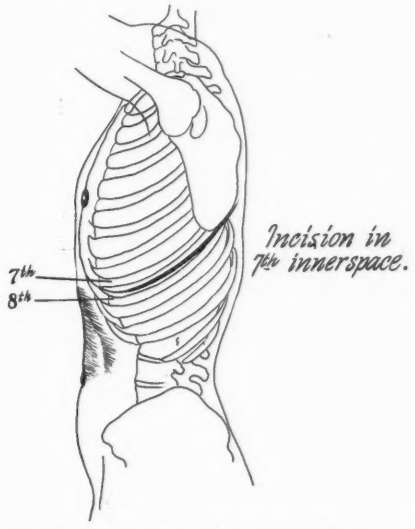

Fig. 5.—Incision in seventh interspace, left, from mid-scapular line to costochondral junction.

THORACIC APPROACH

Endotracheal anesthesia with nitrous oxide, oxygen, and ether is supplemented with curare. The lungs are inflated at intervals of 20 minutes or less while the pleural cavity is open.

An intercostal incision is made in the seventh interspace extending from the midscapular line to the costochondral junction. Sufficient exposure is obtained without coastal resection. Heavy gauze packs are placed between the blades of the rib spreader and tissues to lessen trauma. Pleural adhesions which may be present in the field of operation are divided and the lung is retracted upward with a sponge on holding forceps. Diaphragmatic excursions are inhibited with curare. A longitudinal incision 6 cm. in length is made in

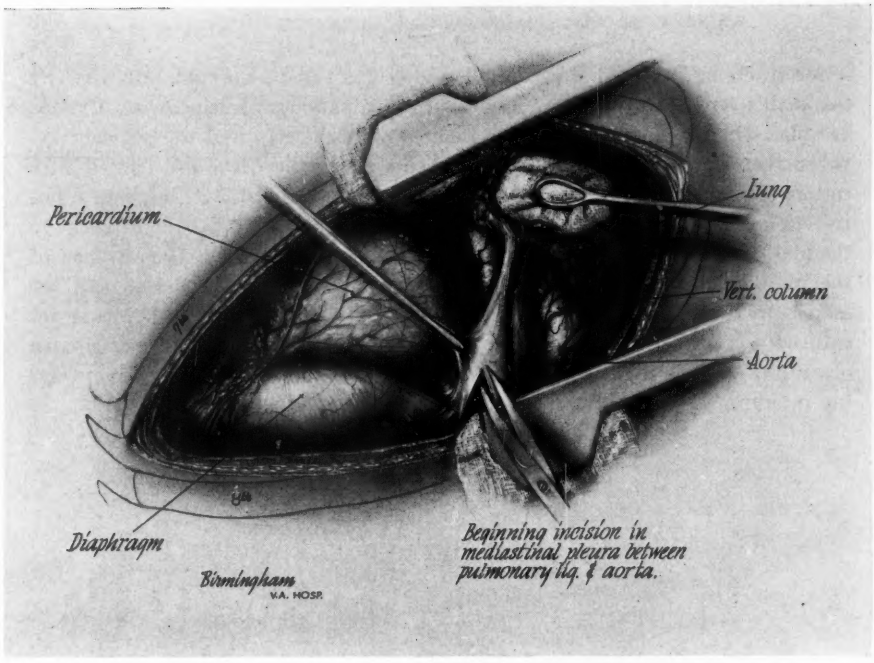

Fig. 6.—Adequate exposure without sectioning rib. Retraction of lung with sponge on holding forceps. Incision in mediastinal plura to expose esophagus.

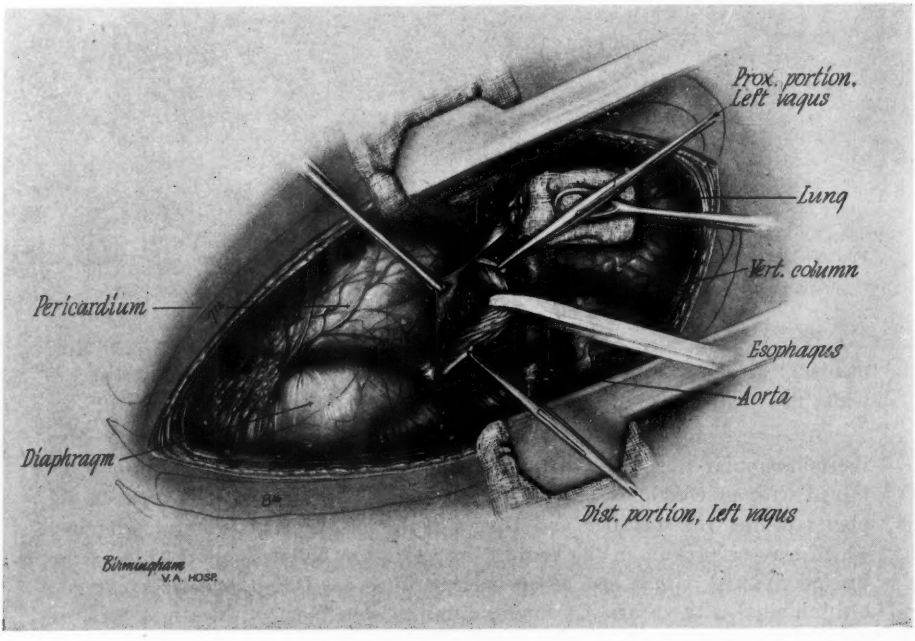

Fig. 7.—Esophagus retracted by gauze tape; segment of vagus nerve resected between ligatures.

the mediastinal pleura between the pulmonary ligament and the aorta, without dividing the pulmonary ligament. The esophagus is separated from its bed by finger dissection and the nerves are identified and dealt with as described for the abdominal approach. The mediastinal incision is accurately closed with interrupted No. 60 cotton sutures. Interrupted sutures are used to unite the incised intercostal muscles together with the parietal pleura. Air is expelled from the pleural cavity by means of a watersealed catheter left in the wound until the last pleural suture is placed. The divided extracostal muscles and skin are closed in layers.

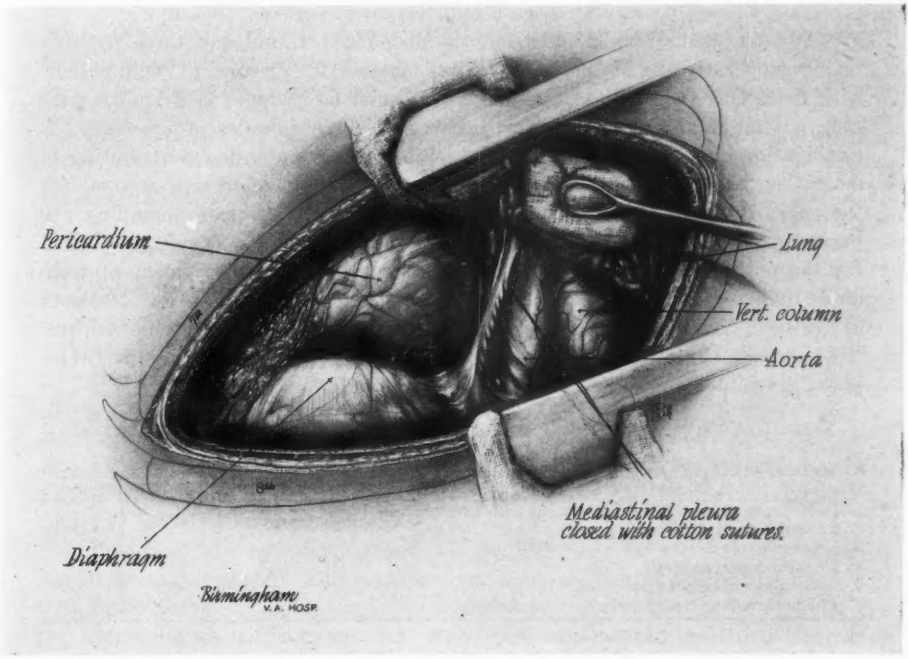

Fig. 8.—Accurate closure of mediastinal pleura with interrupted sutures.

POSTOPERATIVE CARE

The postoperative care is essentially the same for both procedures. The patient sits on the edge of the bed on the afternoon of the day of operation and is gradually ambulatory from then on. Wangensteen suction is used continuously for at least three days and longer in those cases in which severe distention persists. A surgical liquid diet is given on the fifth day and is changed to a surgical soft diet on the eighth day. Water, salt, vitamins and amino acids, sodium chloride, and glucose are given intravenously during the period of intubation. Small frequent feedings with a balanced diet are given as soon as the tube is removed. Patients with gastroenterostomy remain on a liquid

diet several days longer. A general diet is allowed after twelve days, but the patient is admonished not to overload the stomach at any one meal.

COMPLICATIONS

Few of the complications are common to both operations. For this reason, the complications will be discussed separately under each of the approaches.

COMPLICATIONS WITH ABDOMINAL APPROACH

All of the abdominal complications occurred among the first twelve cases and were principally due to faults which have been overcome by improved preoperative and postoperative care and improved surgical technique.

Two patients with dehiscence of the abdominal wound and three patients with postoperative hernia may be grouped together for discussion. One patient with dehiscence, a severe asthmatic patient aged 68 years, was a known poor risk, but operation was imperative because of three episodes of bleeding. A combination of excessive handling of the intestines at operation contributing to distention, and an uncontrollable cough postoperatively, could well account for the rupture of the abdominal wound. In all of the other cases, including one patient aged 69 years, excessive distention, malnutrition, and cough are blamed for the postoperative weakness of the abdominal wall. Improvement of technique by better relaxation, less handling of the intestines, more effective closure of the wound, closer attention to continuous gastric suction, and better preoperative nutrition have done away with the complication in all except the first twelve patients operated upon by the abdominal approach.

TABLE I. POSTOPERATIVE COMPLICATIONS IN THIRTY PATIENTS WITH THE ABDOMINAL APPROACH

COMPLICATION	NUMBER OF CASES
1. Dehiscence of the abdominal wound with evisceration	1
2. Partial wound dehiscence	1
3. Postoperative hernia	3
4. Intestinal obstruction	2
5. Bronchial pneumonia (one death)	2
6. Phlebothrombosis with pulmonary embolism	1

Two patients developed obstruction on the seventh and twentieth postoperative day, respectively. This is probably more than a coincidence. The intestine appears to undergo peculiar segmental contraction in the early days following vagus nerve interruption, possibly due to the sudden removal of vagal control. Attention has been directed to this phenomenon by Alvarez.[2] Unregulated bowel activity and distention are assumed to be the principal factors leading to obstruction in these cases. If this assumption is correct, closer attention to deflation by continuous gastric suction postoperatively should lessen its incidence.

Distention of the stomach and intestines and elevation of the diaphragm are considered to have been factors in the two cases of postoperative pneumonitis. The patient who died, a prematurely aged individual who was considered a relatively poor risk, was overconstricted with a tight abdominal binder, and did not tolerate well the presence of the gastric tube. Since then, the training of

the patient in swallowing and retaining the suction tube before operation has paid dividends in practically doing away with severe postoperative distention.

The patient with phlebothrombosis and pulmonary embolism was heparinized as soon as the complication was discovered and he made a satisfactory recovery.

COMPLICATIONS WITH THORACIC APPROACH

The postoperative complications with the thoracic approach are listed in Table II. Intercostal neuritis occurred in two patients. A segment of rib had been removed in one and a rib was fractured in the other at operation. Severe pain persisted for several months in the first, and for one week in the second. All of the patients with these exceptions have been remarkably free from pain following operation. The absence of intercostal neuritis is credited to the fact that most of the operations were done without costal resection.

TABLE II. POSTOPERATIVE COMPLICATIONS IN THIRTY PATIENTS WITH THE THORACIC APPROACH

COMPLICATION	NUMBER OF CASES
1. Intercostal neuritis	2
2. Pleural fluid (two severe with escape of fluid through wound)	5
3. Pneumonitis	2

Pleural fluid accumulated in large quantity in two cases, neither of which progressed to empyema. One of these patients had a large extrapleural abscess following the injection of Nupercaine in oil in the intercostal space. It is probable that the abscess together with trauma induced by dividing numerous dense pleural adhesions were the cause of the transudate. The practice of injecting the intercostal nerves has since been discontinued. The second patient had a massive accumulation of fluid in the left pleural cavity associated with an extensive bilateral pneumonia. Fluid in the other three cases was minimal and was absorbed spontaneously.

Pneumonitis was bilateral and extensive in one of the two cases and moderate in the other. The more serious case was one of those described as developing severe hydrothorax.

Summing up the complications, there were two cases of intestinal obstruction with the abdominal approach and none with the thoracic, two cases of pneumonia with the abdominal, one resulting in death, against two cases with the thoracic approach. There was one case of phlebothrombosis with the abdominal approach and none with the thoracic approach. Of the complications peculiar to each method, there were two cases of wound dehiscence and three of postoperative hernia of the abdominal wall; and two cases of intercostal neuritis and two cases of large accumulation of serous fluid in the pleural space.

CLINICAL RESULTS

The period of observation in both groups of cases is too brief to make an evaluation of the effectiveness of vagus nerve section in healing peptic ulcers. However, a review of the early results will aid in making a comparison of the merits of the two methods. In the comments which follow, all cases in which there has not been complete relief are counted as failures.

There was one death with the abdominal approach, due to postoperative pneumonitis. Fifty-six of the sixty patients undergoing vagus nerve section have experienced complete symptomatic relief. It should be taken into consideration that fifteen of the sixty patients had a gastroenterostomy performed in addition to the denervation. In four, the anastomosis was performed subsequent to vagus nerve section because of persistent pyloric obstruction.

There were two failures with the abdominal approach. They are reported here.

S. H., aged 40 years, had a vagus nerve section for treatment of a gastrojejunal stomal ulcer. After the operation he believed himself to be only "50 per cent relieved" and he still has frequent attacks of epigastric pain and vomiting. The acidity, which was not high before operation, remains unchanged. Roentgenologic examination has not been informative.

H. C. was a patient aged 42 years. An inflammatory lesion at the junction of the pylorus and duodenum was found at operation. Exposure was poor and the hiatal region was not well visualized. The cardia of the stomach was inadvertently opened during the finger dissection and was closed with intestinal suture. Two nerves, supposedly the right and left vagus nerves, were divided, but examination for other nerve segments was unsatisfactory. One year after operation there had been no improvement in the patient's condition.

There was one failure with the thoracic approach. The case is described here.

R. G., aged 36 years, began to have pain two months following discharge which was identical with that experienced before surgery. Studies of the insulin test and gastric motility indicated that vagal denervation of the stomach was incomplete.

Thus, there are three failures in the series; two with the abdominal approach and one with the thoracic. These are ascribed to incomplete denervation.

One case of erroneous diagnosis is worthy of special mention because of its bearing on the choice of operative approach. The patient had undergone a pyloric antrum resection elsewhere six weeks previously and experienced pain and hemorrhage following operation. Roentgenologic examination indicated a probable stomal ulcer but the evidence was inconclusive. Abdominal exploration revealed a localized abscess lying between the enterostomy and the transverse colon. Drainage of the abscess resulted in complete relief. Had the thoracic approach been used and vagotomy performed, the patient would have been classed as a failure, and it is even possible that a gastrojejunocolic fistula might have been formed.

In our early cases the operation was so much easier to accomplish through the thorax that we felt it should be used except in instances of pyloric obstruction, obscure diagnosis, malignancy either proved or suspected, or other abnormalities either known or suspected. However, with more experience and improvement in technique it was found that the denervation could be accomplished as satisfactorily with the abdominal approach, and we now use that method routinely. The advantages of being able to observe the lesion and perform gastrojejunostomy far outweigh the difficulties inherent in the abdominal approach.

SUMMARY AND CONCLUSION

This early report on the results of vagus nerve section for the treatment of peptic ulcer in sixty patients is concerned chiefly with determining the relative merits and the indications for thoracic and abdominal approaches. In our earliest cases the operation was easier to accomplish and the complications were less severe and less frequent when the operation was performed through the thorax. As experience increased objectionable features of the abdominal approach were overcome and we now perform the operation routinely through the abdomen because of the advantages of being able to examine the lesion and perform gastrojejunostomy at the same time.

Performance of the operation of vagal denervation of the stomach should be preceded by a thorough acquaintance with the patterns and relative anatomy of the vagus nerves in the lower mediastinum.

An analysis of the results in our sixty cases suggests that failure to accomplish relief by vagus section should be ascribed to failure to perform the operation properly, or to faulty diagnosis.

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EXPLORATORY T..ORACOTOMY IN THE MANAGEMENT OF PLEURAL EMPYEMA

PAUL W. SANGER, M.D.
CHARLOTTE, N. C.

THE incidence of empyema in the pleural cavity has decreased since the advent of chemotherapy and the end of World War II. Empyema following lobar or lobular pneumonia is now rarely seen. Thus, empyema from hitherto unusual etiologic conditions is more commonly encountered in the practice of thoracic surgery. That which necessitates surgical treatment usually constitutes a complication of such conditions as bronchiectasis, trauma of the chest not associated with hemothorax, infected hemothorax, ruptured lung abscess, and ruptured subdiaphragmatic abscess, and as a consequence of tube drainage or too small rib resection in treating postpneumonic empyema.

I have the greatest respect for the time-tested surgical principle of draining an intrapleural abscess by tube insertion or rib resection; although in dealing with the type of case described here, such treatment alone in my experience usually has been inadequate.

Decortication of the lung has been recognized as sound management in acute, massive, or multiloculated pleural abscesses.[1] The mode of attack in present-day empyema, however, is being altered with the change in frequency of its underlying cause. It is now believed that an exploratory thoracotomy is indicated in any case of acute or subacute empyema of the pleura that does not satisfactorily respond to chemotherapy, aspiration, closed drainage, and general supportive measures. By means of this exposure it may be determined in each individual case which procedure to follow; whether decortication of the lung suffices, further surgery is indicated then, or surgery should be delayed for a subsequent operation. Illustrative cases will be presented and the problems and indications discussed for the surgical procedures which were chosen in each instance.

An exporatory thoracotomy can be done with safety only if the following conditions are fulfilled: (1) restoration and continuance of whole blood; (2) administration of large amounts of penicillin or streptomycin, depending upon the infecting organisms; (3) the availability of a competent anesthetist. With these protections almost no patient is too ill to withstand pulmonary surgery.

Each case is individualized in its management but comments will be made upon a few points of procedure. The trite but so important history cannot be overemphasized in trying to ferret out symptoms of a chronic pulmonary ailment such as productive cough and other symptoms suggestive of a pre-existing bronchiectasis. Roentgenograms of the chest are confined to anteroposterior and lateral views since stereoscopic films of the chest, in my opinion, are misleading.

From the Surgical Service of Charlotte Memorial Hospital.
Read (by title) at the Twenty-seventh Annual Meeting of The American Association for Thoracic Surgery, St. Louis, Mo., May 28, 29, and 30, 1947.

The chest fluid is aspirated and examined bacteriologically, preferably prior to antibiotic or chemotherapy. If this is not possible, para-aminobenzoic acid or penicillinase, respectively, is added to the culture media.

Hematologic examinations invariably include hematocrit readings. Blood transfusions are given until the hematocrit reading has reached a level of 50 or above. An intercostal tube may be placed into the empyema pocket and connected to a water-sealed jug resting upon the floor.

This latter procedure, however, is merely preliminary to the definitive treatment which follows after the cell volume has been restored to normal and after bronchography and bronchoscopic examinations in certain cases have been made. Bronchograms of the involved side are not omitted when there are suspected pulmonary communications into the pleural cavity or when there is suspected bronchiectasis which may warrant extirpation of the lobe or segment involved.

Under endotracheal anesthesia and continuous intravenous blood infusion an exploratory thoracotomy is undertaken. It is important that the infusion should be given simultaneously into two veins in order to sustain adequate blood volume. If the empyema is extensive, it is explored routinely through the fifth or sixth rib bed; otherwise, it is opened through a selected rib bed directly over the pocket. The pus is evacuated and decortication is carried out as previously described.[1] The lung is re-expanded if possible but when extensive bronchiectasis exists or when a lung abscess or malignancy is demonstrated, it may be advisable to delay extirpation surgery to a later date. In certain instances, however, it is preferable to remove the involved lung or segment in one operation.

TYPICAL CASES OF EMPYEMA RESULTING FROM BRONCHIECTASIS

CASE 1.—B. P., a white, woman patient, 21 years old, was admitted to the hospital on Jan. 18, 1946, with the chief complaint of a sudden onset of a severe, knifelike pain in the left side of the chest, of forty-eight hours' duration, associated with chills, fever, and dyspnea. She had a history of having had bronchiectasis in the left lower lobe of several years' duration. The diagnosis had been established previously in another hospital.

On examination she appeared mildly cyanotic, acutely ill, and in marked respiratory difficulty. The temperature was 103.6° F., pulse was 120 beats per minute, and respirations were 34 per minute. The trachea was grossly displaced to the right (Fig. 1, *A*), the maximum impulse of the heartbeat was to the right of the sternum, and there was dullness throughout the lower left side of the chest and hyperresonance over the apex.

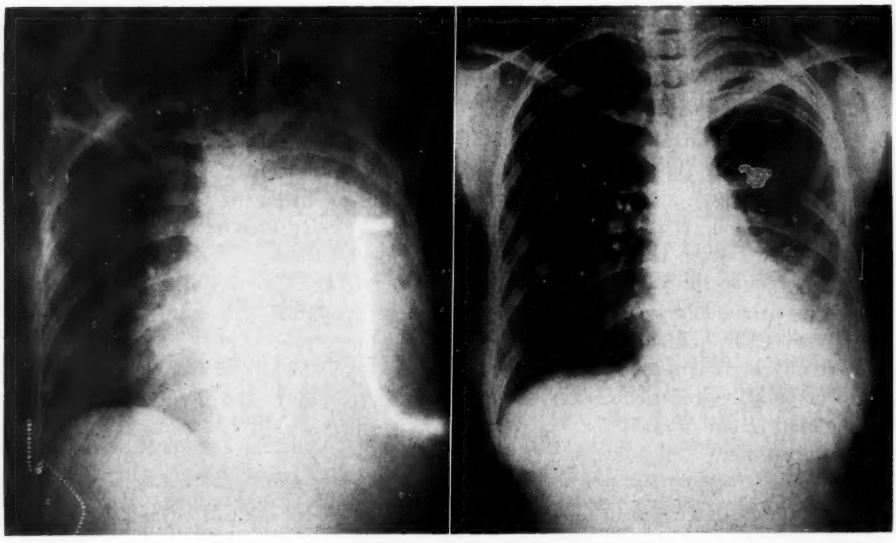
A catheter was placed in the eighth left interspace in the posterior axillary line. A turbid fluid was drained, from which alpha streptococcus and nonhemolytic *Staphylococcus aureus* were cultured. Penicillin, 50,000 units, was administered intramuscularly every three hours. The hematocrit reading was 41 and the patient was given a transfusion of 500 c.c. of blood. The temperature ranged from 101 to 104.6° F. The catheter did not drain satisfactorily and it was reinserted at another site where it also ceased to drain after a few hours. The patient's condition was not improved. Bronchoscopic examination was essentially unrevealing.

On the third day after admission, an exploratory thoracotomy was performed, excising the left sixth rib, under endotracheal anesthesia. Multiloculated empyema pockets and organized fibrin were found upon entering the pleural cavity. A thick capsule incarcerated

the entire lung. The pus was evacuated and the lung was decorticated of its constricting sheet, after which it re-expanded satisfactorily.

In view of the fact that the lobe had re-expanded, it was thought undesirable at this time to proceed with lobectomy. A No. 32 mushroom catheter was placed in the eighth left interspace in the posterior axillary line and connected to a water-sealed jug. The patient received 1,500 c.c. of whole blood and 1,000 c.c. of glucose intravenously during the operative procedure. Bronchoscopic aspiration was done before the patient returned to her room.

Large doses of penicillin were continued. Ambulation was started the following day and the drainage tube was removed on the third postoperative day. On the fifth day the patient was afebrile and so remained during the subsequent stay in the hospital. She was discharged eleven days after the operation (Fig. 1, *B*).



A. *B.*

Fig. 1.—*A*, Note the displacement of the mediastinum to the right as air and fluid in the left have collapsed the left lung. *B*, Ten days after decortication note the re-expansion of the left lung, except for a small area in the apex, and normal position of the mediastinum.

She returned three months later for lobectomy. When this was done, surprisingly few adhesions between the visceral and parietal pleura were found, although the lobes were firmly adherent. The postoperative course this time was without mishap and she was discharged from the hospital ten days after operation.

Comment.—The history of pre-existing bronchiectasis and the sudden onset of the patient's illness with pain, dyspnea, and toxicity caused one to assume that a rupture of a bronchiectatic abscess had occurred. Tube drainage failed to reduce the symptoms and the temperature rose to 105° F. A thoracotomy was then performed. An incarcerated lung was liberated by decortication and the lung was inflated. Therefore, it was thought wise to delay resection for a later date whereby the extent of the disease could be demonstrated more accurately. The lower lobe was removed three months later. The patient, now symptom-free, is with her soldier husband in Alaska.

A case of similar clinical record was found on Dr. Edward Churchill's service at the Massachusetts General Hospital. This individual was treated by tube drainage but a pneumothorax developed which collapsed the lung. It is interesting to note the lack of organized adhesions that should have prevented this collapse. It has been suggested that early administration of penicillin may have played some role in nature's not forming normal adhesions.[2] However, I have not observed any lack of organized fibrin even though some patients had had large quantities of penicillin immediately after the onset of their illnesses.

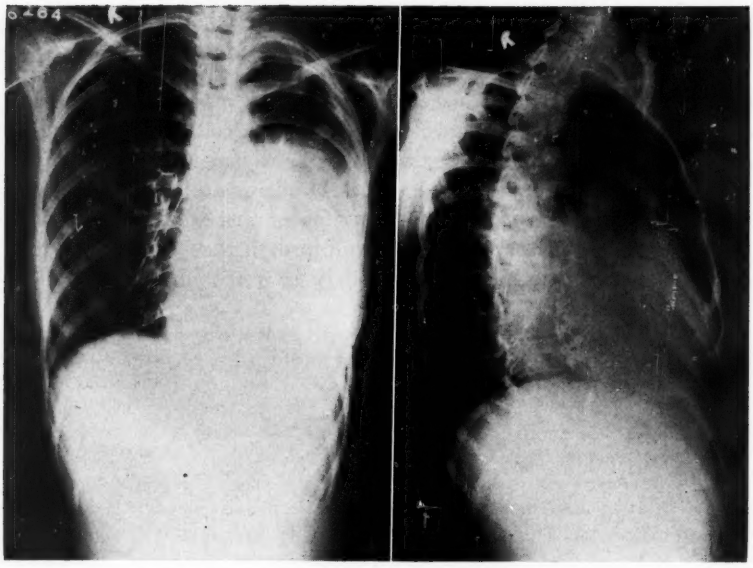
CASE 2.—R. G., a 16-year-old, white female, was admitted to the hospital on Jan. 2, 1947, with the chief complaint of chills, fever, and a persistently draining sinus in the left side of the chest of three months' duration. She had a history of intermittent cough and fever, diagnosed as "bronchitis" in early infancy. Two years before admission she had "lobar pneumonia." In the fall of 1946 she had been hospitalized six weeks after a sudden attack of pleural pain followed by empyema, for which a rib resection drainage had been done. Temporary improvement had occurred, although the cough had persisted with elevation of temperature, and pus had continued to drain from the thoracic sinus.

Three months later she was transferred to the chest service. At this time the temperature was 102.4° F., pulse rate was 110 beats per minute, and respirations were 24 per minute. She appeared chronically ill, underweight, anemic, and apprehensive. Further examination showed that the trachea was in midline and that there was a lag on inspiration over the left side of the chest. There was signs of consolidation over the entire left side of the chest with the exception of the apex (Fig. 2, *A*). A draining sinus in the left posterior axillary line in the seventh rib area was seen. Cultures from this exudate showed a mixed infection of alpha and gamma streptococci, *N. catarrhalis, Diplococcus pneumoniae,* and nonhemolytic *Staph. aureus.*

Since the hematocrit reading was only 30, she was given 1,000 c.c. of citrated blood. Lung mapping demonstrated bronchiectatic changes in the entire left lower lobe, lingula, and base of upper lobe (Fig. 2, *B*). Penicillin aerosol (100,000 units diluted in 2 c.c. of saline solution) was carried out three times daily for three days. Bronchoscopically there was no obstruction to the left bronchus; only excessive exudate was seen.

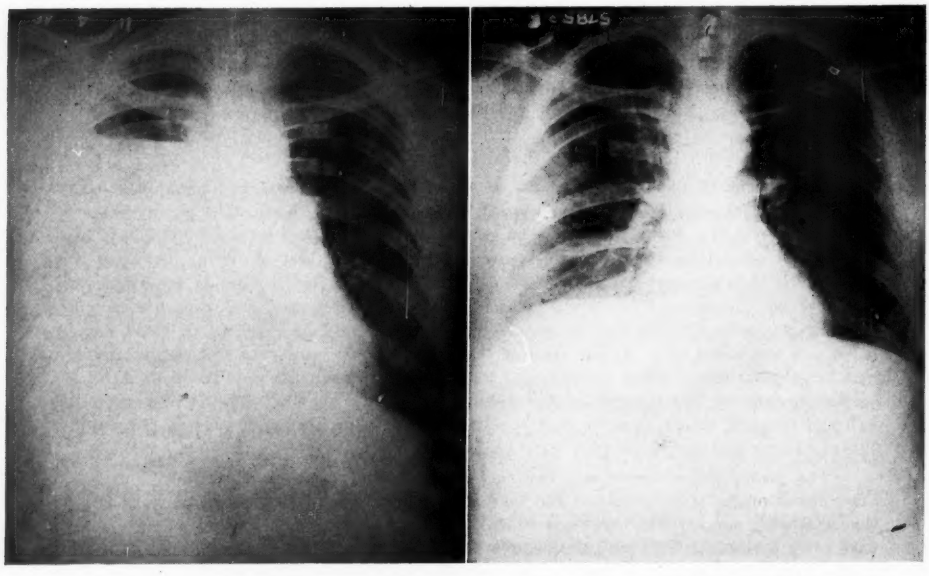
On the fourth day after readmission the patient was prepared for an exploratory thoracotomy. The old sinus tract was excised in exposing the sixth rib. The pleura was very thick and its cavity was divided into pockets filled with purulent exudate. The lung was found to be constricted by a very thick, organizing, vascular sheet of fibrin. The lung was freed of its imprisonment but the lobes would not re-expand due to chronic atelectasis and bronchiectasis. Because of this it was determined necessary to proceed with a pneumonectomy. Bleeding was quite a factor but the blood volume was sustained by 2,000 c.c. of blood flowing in an arm and ankle vein. At the close of this procedure the chest was thoroughly irrigated with large quantities of saline solution, and 100,000 units of penicillin were instilled. A No. 30 mushroom catheter was inserted in the eighth left interspace. She withstood the operation well and returned to her room in good condition. The following day the hematocrit was 54. The customary postthoracic surgical care was given.[3]

The postoperative course was uneventful and recovery was rapid. Twenty-four hours after operation she was out of bed and the catheter was removed eighteen hours later. At no time following surgery did the temperature rise above 99.6° F. She was discharged eleven days after operation. This patient was seen four weeks later at which time she was afebrile, free of cough, and had gained weight. It was expected but, peculiarly enough, not until two months after pneumonectomy did this patient develop signs and symptoms of pleural infection; but the wound was uninvolved. The pocket, which was relatively small, was successfully collapsed by a one-stage thoracoplasty.



A. *B.*

Fig. 2.—*A*, This shows an area of density on the left that reaches to the second interspace anteriorly and a clear right lung with normal lung mappings. *B*, Semi-oblique views are more informative in revealing bronchiectatic changes with lipiodol in the presence of pleural exudate.



A. *B.*

Fig. 3—*A*, This film reveals an area of density that occupies the majority of the right pleural cavity with fluid level at the first interspace anteriorly. The remainder of the right chest cavity is filled with air. There is no noticeable displacement. *B*, This discloses satisfactory re-expansion of the entire right lung with an elevated diaphragm. Patient was symptom-free and returned to work four weeks later.

Comment.—Recognizing the rarity of ordinary empyema, an attempt is made to be alert to the unusual origin of this disease. Previously a bronchoscopic examination had been done but it failed to disclose any pathology. The referring physician surmised bronchiectasis when the cough persisted and improvement did not follow drainage of the empyema pocket. This case illustrates the justification of using lipiodol though we take the risk of creating or aggravating pneumonitis. It is also pertinent to note the extensive surgery done "through pus" without resultant acute pleural infection which may have been unwise, but it was felt that "our hand was called" when expansion of the lung after decortication was unsuccessful. The criteria in determining the need for resection of the part involved is whether or not the lobe or segment will expand under positive pressure or deflate with the release of same.

In that this youngster had already run the gamut of conservatism, it was felt justified to try to complete the surgery in one operation although weeks later the pleural space did become clinically infected. A remarkable improvement has been that of her mental attitude, which is important at the age of 16 while entering adulthood. She is now symptomless two months after thoracoplasty.

EMPYEMA AS COMPLICATION OF SUBDIAPHRAGMATIC ABSCESS

CASE 3.—C. C., a 23-year-old former sergeant, was admitted to the hospital by ambulance on Oct. 31, 1946, with the chief complaint of fever and pain in the right side of the chest, associated with generalized malaise. He had a history of persistent diarrhea, with stools containing blood and mucus, of two months' duration in 1943 while on military duty in Africa. With the exception of dysentery, he had no illness until approximately six weeks prior to admission when he suffered "an attack of pneumonia," involving the right lung. He apparently responded well to chemotherapy for three weeks, after which he developed intermittent fever associated with "a tired, worn-out feeling." Shortness of breath on exertion and a slight, nonproductive cough were also complaints.

When examined, he was a well-developed white man, acutely ill, listless, and moderately short of breath. Temperature was 102.6° F., the pulse rate was 108 beats per minute, and respirations were 28 per minute. The trachea was to the left since the right chest did not expand normally on inspiration. There were signs of air in the right apex and fluid in the right base. The heart was not remarkable. The left lung fields appeared clear. It was impossible to palpate the liver, although there was tenderness to fist percussion in the liver area. The muscles were somewhat splinted in the right upper quadrant but no rebound tenderness was noted. No viscera or masses could be felt. The laboratory findings showed a red cell count of 3,800,000 per cu. mm. and a white cell count of 20,200 per cu. mm. The hematocrit reading was 36.

The right side of the chest was aspirated and 500 c.c. of tenacious, anchovy-like material were withdrawn. Routine smears and cultures failed to reveal any organisms but numerous pus cells were found. X-ray examination of the chest, anteroposterior and lateral views, showed a hydropneumothorax on the right side with two fluid levels and a slight shift of the mediastinum to the left (Fig. 3, *A*).

Whole blood, 1,000 c.c., was given immediately and Demerol, 50 mg., was needed repeatedly for the control of pain in the chest. Further therapy consisted of 50,000 units of penicillin every three hours. The stools were negative for *Amoeba histolytica* as were the cultures and smears from the chest fluid. Bronchography showed no bronchiectasis or bronchial communication through the diaphragm. A preoperative diagnosis was made of empyema resulting from a ruptured subdiaphragmatic abscess thought to have been secondary to abscess of the liver, questionably of amoebic origin.

An exploratory thoracotomy was done through the right sixth rib bed on the third day after admission. The pleural cavity was found to be filled with a purulent, nonodorous, pasty, brownish exudate. After decorticating and re-expanding the lung, no bronchopleural fistula could be demonstrated though the communication through the diaphragm was easily identified. This led to a subdiaphragmatic and liver abscess. Through another incision, a segment of the right eleventh rib was resected and an extrapleural communication was made into the subdiaphragmatic abscess cavity into which two cigarette drains were inserted. Thereupon the perforation of the diaphragm was débrided and closed with interrupted silk sutures. The patient withstood the operation very well. The systolic pressure did not fall below 90 mm. Blood, 2,000 c.c., was given to sustain the patient during this surgery. Two large mushroom catheters were inserted in the chest cavity, one in the second intercostal space in the anterior axillary line and the other in the eighth interspace in the posterior axillary line. Suction was applied to the anterior catheter for a period of twenty-four hours, after which it was removed. The posterior catheter was irrigated daily for five days, then removed.

Routine care was followed, which in this case necessitated intercostal nerve blockings on three occasions. Emetine therapy was begun. On the fourth postoperative day the patient developed some asthmatic symptoms which were relieved by aminophylline suppositories.

The patient was afebrile on the third postoperative day and remained so until discharge from the hospital fourteen days after operation. Examination two months later showed a satisfactory re-expansion of the right lung (Fig. 3, *B*). The patient returned to his duties as cotton mill worker nineteen days after discharge from the hospital.

Comment.—In this individual, by having a large thoracotomy opening it was possible to estimate accurately the extent of the damage, decorticate the lung (thereby re-expanding it), and more easily drain the subdiaphragmatic abscess as well as close the communication between the chest and the abdomen. Such could not have been accomplished unless there was a large thoracotomy opening. Hospitalization was reduced many days as the patient returned to his former occupation thirty-three days after operation.

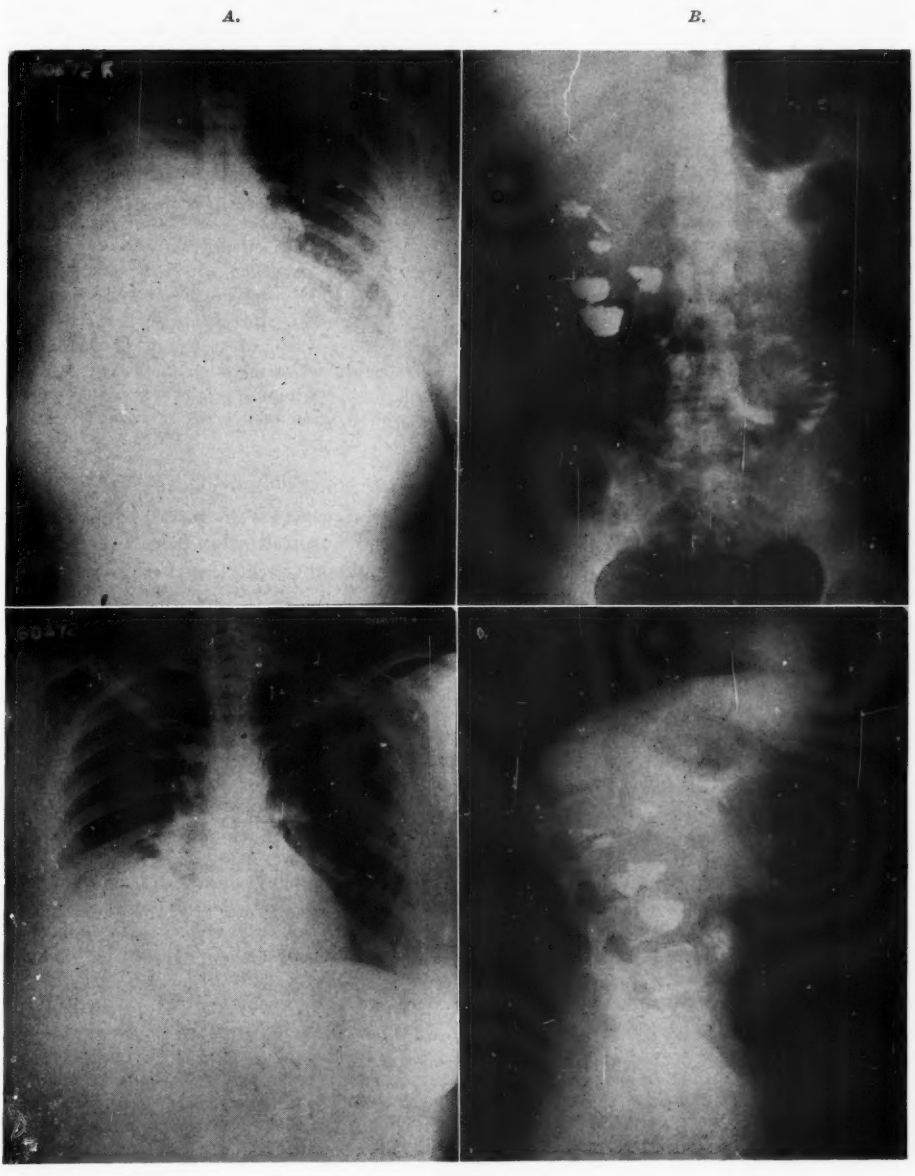
BRONCHIAL-RENAL FISTULA

CASE 4.—E. B., a 31-year-old white housewife, was seen in consultation on her second admission to the hospital when she was complaining of chills, fever, and a productive, foul cough of six days' duration.

On the previous entrance two months before there had been a diagnosis of right renal calculus, pyelonephrosis, and empyema in the right chest (Fig. 4, *A*). She was treated with repeated chest aspirations, penicillin, and other supportive measures. On this regime the patient improved. After eighteen days she went home for further improvement preparatory for a kidney operation.

The course at home was indifferent until ten days before admission, when a severe pain in the right shoulder region developed, associated shortly afterward with chills, fever, and paroxysmal attacks of coughing. The cough soon became productive with copious quantities of foul-smelling, reddish material. Coincidental with this the pain in the right shoulder disappeared although the chills, fever, and cough continued. The latter was made worse when she lay on the left side. The temperature ranged up to 104° F., pulse was 120, respirations 22, white blood cell count 18,000, and hematocrit 36.

On further examination she did not appear as ill as her vital signs disclosed. There was a slight lag on the right on inspiration. Breath sounds were distant throughout the right side of the chest. The percussion note also was impaired. Dullness in the right base was thought to be due to an elevated diaphragm. There was moderate tenderness in the kidney region but no tenderness over the liver could be elicited.



A. *B.*

C. *D.*

Fig. 4.—*A*, Anteroposterior view shows a homogeneous blurring of the lower two-thirds of the right pleural cavity. Empyema in this instance was secondary to subdiaphragmatic abscess. *B*, Bronchograms and injection of lipiodol into abscess cavity disclosed "puddling" of the opaque substance, which indicated a bronchial-renal fistula and at operation disclosed communication into a pylonephrotic kidney. *C*, Shows satisfactory clearing of the right lung although the diaphragm remains elevated. Four weeks after drainage of the subdiaphragmatic abscess and nephrectomy, the bronchial fistula had closed and patient was free of all respiratory symptoms.

There was demonstrated a bronchopleural communication beneath the diaphragm as evidenced by the same character of fluid there obtained as the patient coughed; and by bronchograms and lipiodol injections it was possible to demonstrate "puddles" of the opaque substance below the diaphragm in the region of the right kidney (Fig. 4, *B*).

Bacteriologically, gram-negative rods and alpha streptococci were cultured from the sputum and aspirated exudate. The patient was given 1,000 c.c. of whole blood prior to surgery. Six days after admission the eleventh and twelfth ribs were resected subperiosteally through which a large abscess cavity was encountered in the subdiaphragmatic area. This cavity was partly filled by a large, ruptured, pyelonephrotic, right kidney. After decompressing the abscess the diaphragmatic fistula could not be identified. The kidney was removed in a subcapsular manner.* Cigarette drains were inserted and the wound closed in anatomic layers. During the operation the patient received four pints of blood and 1,000 c.c. of glucose.

The postoperative course was satisfactory. The temperature did not exceed 100.4° F. Early ambulation was started and the patient was discharged two weeks afterward with a small No. 14 catheter in place. Roentgenograms then showed an elevated diaphragm but relatively clear chest cavity. The cough had stopped and there were no symptoms suggestive of the bronchial fistula being open. The patient was seen again four weeks after operation and she was free of respiratory symptoms (Fig. 4, *C*).

Comment.—Pleural infection and a diaphragmatic bronchial fistula, secondary to a pyelonephrotic kidney, cause one to respect the bizarre origin of present-day empyema. Would the morbidity and hospitalization have been appreciably reduced if an exploration had been made on the first admission to the hospital two months previously?

TRAUMATIC EMPYEMA

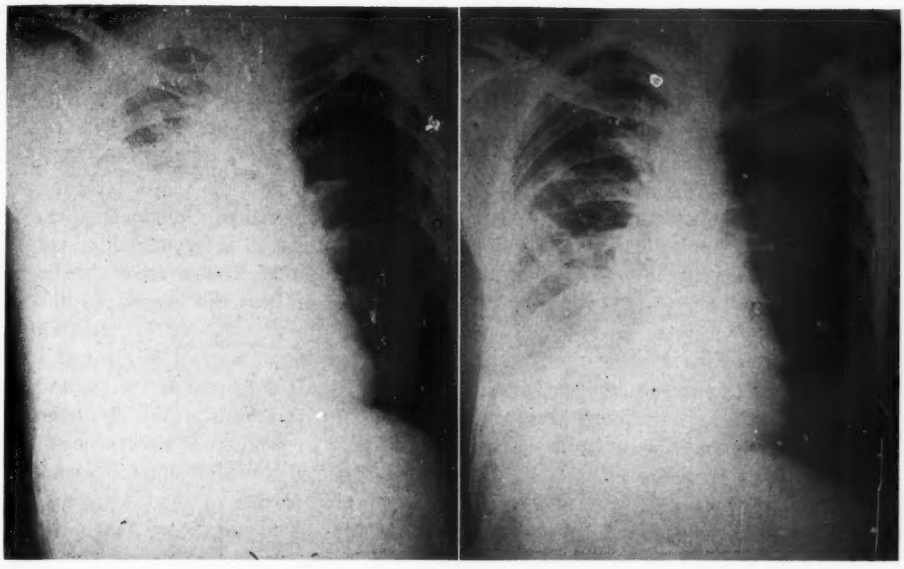
CASE 5.—F. H., a 32-year-old white man, was admitted to the hospital on Jan. 2, 1946, because of increasing difficulty in breathing, with chills and fever of twelve days' duration. He gave the story of having "been in a fight and sustained a knockout blow on the right chest" some seventeen days previously. Apparently no evaluation of intrathoracic involvement had been made at that time.

At physical examination he appeared acutely ill and rather pale. Breath sounds were absent, percussion note was dull over the posterior base of the right chest, and no scars were seen (Fig. 5, *A*). Aspiration obtained a thick, turbid material that grew a mixed culture of cocci and bacilli. Temperature was 103° F., pulse rate 116 beats per minute, and respirations were 30 per minute. Penicillin in large doses had been given prior to admission and this was continued in 50,000 units every three hours. As the hematocrit reading on admission was 30, he was given 1,500 c.c. of whole blood. Repeated attempts at aspiration of the chest obtained a very small amount of thick pus. Temperature ranged from 101 to 105° F. The white blood cells numbered 21,000 per cu. mm. The urinalysis was negative.

An exploratory thoracotomy was done on the right side with resection of the seventh rib. The chest cavity was filled with organizing, foul-smelling exudate. The posterior and lateral portions of the lower lobe were partially compressed, being held with a constricting sheet of exudate. This was removed, thus allowing the lung to re-expand and fill the previously existing empyema cavity. A No. 20 mushroom catheter was placed in the eighth interspace in the posterior axillary line. The patient received 2,000 c.c. of whole blood during the operation. Penicillin therapy was continued.

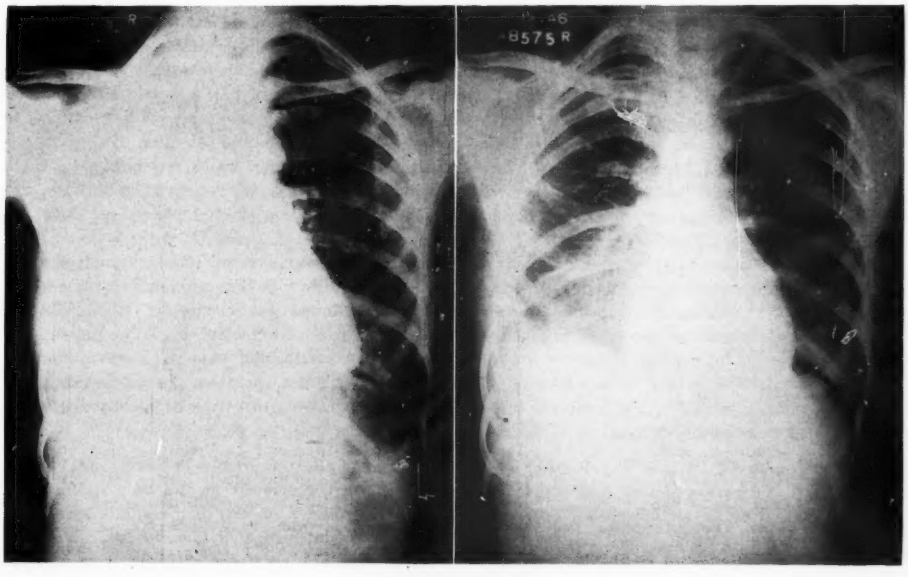
Progress was uncomplicated. The temperature subsided on the fifth postoperative day to normal and so remained. The drain was removed seventy-two hours after operation. The patient was discharged with the lung re-expanded and in good condition on the tenth day after surgery (Fig. 5, *B*). He has remained free of respiratory symptoms to date.

*By Dr. Roberts McKay.



A. *B.*

Fig. 5.—*A*, There is an isolated empyema pocket localized in the posterolateral region which was explored through the right sixth rib. The cavity was evacuated and the lung was decorticated. *B*, This shows very satisfactory re-expansion of the lung and elimination of the empyema ten days after operation.



A. *B.*

Fig. 6.—*A*, The right lung is obliterated, which proved to be caused by organized, foul, infected hemothorax. *B*, Fourteen days after operation the right lung is re-expanded. The semi-density along the margin of the pleura is interpreted to be that of organized fibrin on the parietal pleura, which was not thought necessary to remove at the time of decortication.

Comment.—The presumptive cause of this patient's empyema was a traumatic, infected, organized hemothorax. Such being the case a bronchoscopic examination and bronchograms were not indicated, although at operation no evidence of organized hemothorax was apparent. Here we were dealing with a localized empyema pocket and by making the incision longer and excising slightly more rib than usual, the cavity was emptied of its pus. Under visualization, with adequate exposure the lung was decorticated and re-expanded, which allowed the cavity to be filled with a useful, healthy lung. The intercostal tube was removed three days after operation. The patient was discharged on the tenth postoperative day. This is in contrast to the usual way of treating empyema pockets, that is, insertion of a tube or resection of a small segment of a rib. The majority do recover, but such a patient treated by the latter method usually remains in the hospital more than two weeks with uncomfortable drainage of pus from the tube or sinus. Furthermore, daily dressings are always necessary. Aside from the economical, physical, and mental considerations, it seems to be false conservatism to prefer an inadequate exploration of these empyema cavities. In short, it is felt that this case gives support to the theory that more extensive surgery in individuals with large or even small empyema cavities is actually the conservative measure.

INFECTED ORGANIZED HEMOTHORAX

The next two cases represent clear-cut problems.

CASE 6.—C. S., a 48-year-old, white woman, was admitted to the hospital on Feb. 25, 1946, complaining of pain in the anterior right part of the chest following a stab wound three weeks previously. The patient was slightly dyspneic and the temperature ranged from 101.6 to 104.6° F. The pulse was rapid, around 112 to 120 beats per minute. She was anemic, with a red blood count of 2,900,000 cells per cu. mm. and a hematocrit reading of 31. Pertinent findings of the right chest were dullness to percussion and absence of breath sounds (Fig. 6, *A*). A thoracentesis yielded a dark, thick, bloody fluid which was negative on smear, although it had a putrid odor. No organisms, however, were grown on culture. Penicillin had been given previously. It was reinstituted and she was given 1,200 c.c. of whole blood.

On the second day after admission, an exploratory thoracotomy was done through the right sixth rib and there the chest was found to be filled with an odorous, organized, infected hemothorax. The lung was decorticated and allowed to re-expand. A No. 32 catheter was placed in the eighth interspace and connected to a water-sealed jug. The patient had an uneventful postoperative course and was out of bed twenty-four hours after operation. She was afebrile on the fifth postoperative day. Ten days after operation the patient was discharged (Fig. 6, *B*). She returned to her work as a seamstress three weeks later and has remained well since that time.

CASE 7.—W. M., a Negro man, 40 years old, was admitted to the hospital on Feb. 10, 1946. He gave a history that approximately nineteen days previously he had been stabbed from behind by an unknown assailant. There had been shortness of breath immediately and profuse bleeding. The wound had been sutured eight hours later in a distant hospital. Presumably the wound did suck. The patient's physician aspirated 1,500 c.c. of blood from the left side of the chest, and in subsequent aspirations 600 c.c. and 1,000 c.c. were drawn on successive days. After that no further fluid was obtained and he was given no supportive blood, although he did receive penicillin. For nineteen days the patient remained short of breath. Chills and an elevation of temperature which ranged around 104° F. developed.

When admitted to our hospital he was poorly nourished, pale, dyspneic, and perspiring freely. The trachea was in the midline. The chest was symmetrical with a definite lag on the left. The right lung field was relatively clear. The left was dull to percussion over its entirety. The abdomen was peculiarly spastic and tender in the left upper quadrant; however, there was no rebound tenderness. The hematocrit reading was 28. Hemolytic staphylococcus and *Bacillus coli* were cultured from the aspirated fluid. X-rays of the chest (Fig. 7, *A*) showed homogeneous density over the entire left lung field. The lateral film showed air anteriorly and a fluid level posteriorly. Aspiration obtained 1,500 c.c. of air and only 500 c.c. of purulent, offensive, bloody material were drawn from the left posterior eighth interspace.

The patient was given 1,000 c.c. of blood preoperatively and prepared for an exploratory thoracotomy which was done two days afterward through a section of the left sixth rib. The lung was encased and the thoracic cavity filled with an organized, infected hemothorax. Decortication was carried out in the usual manner. Bleeding was quite annoying. The patient was supported on 1,500 c.c. of blood and 1,000 c.c. of plasma. Two catheters were used, one in the anterior second interspace and one in the posterior axillary line in the eighth interspace.

He had an uneventful postoperative course and was allowed out of bed on the second day. The catheters were removed on the second and fourth days, respectively, after the operation. He was afebrile forty-eight hours postoperatively and so remained until discharge eight days after surgery (Fig. 7, *B*). He returned to his occupation as a chauffeur three weeks after leaving the hospital.

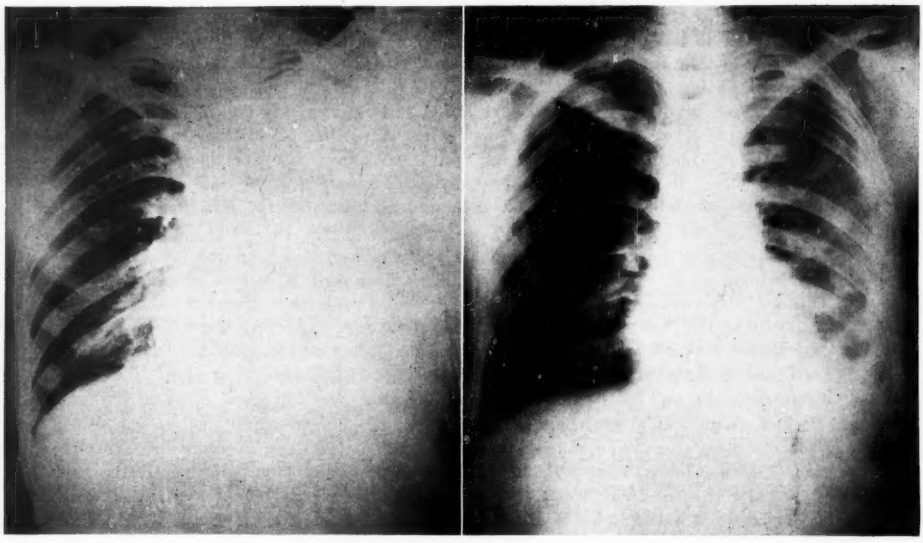
Comment.—These two cases are typical of the badly infected, organized hemothorax which notably is the type that experience in World War II taught us could be decorticated with impunity[4]; otherwise the patient would likely become a pulmonary cripple unless the lung was freed, thus allowing it to re-expand and fill the pleural cavity. It is a matter of record that these soldier patients, for the most part, were readily restored to useful military duty within a period of a few weeks.

PNEUMOPYOTHORAX AS RESULT OF INADEQUATE SURGERY IN POSTPNEUMONIC EMPYEMA

CASE 8.—J. M., a 40-year-old, colored man, was seen at the hospital in October, 1946. The chief complaint was a productive, foul cough, "lung abscess," and an abdominal hernia of ten months' duration. He gave a story of having had a "gall bladder operation" ten months previously. During this stay in the hospital he developed pain in the left chest, shortness of breath, and an elevation of temperature. A diagnosis of pneumonia on the left side, complicated by empyema, was made. The old record showed that the chest cavity was drained by rib resection and insertion of a tube, but his shortness of breath and pain in the chest with drainage of excessive pus, chills, and fever continued after discharge from the hospital. Soon after going home he developed a large postoperative hernia in the right upper rectus incision.

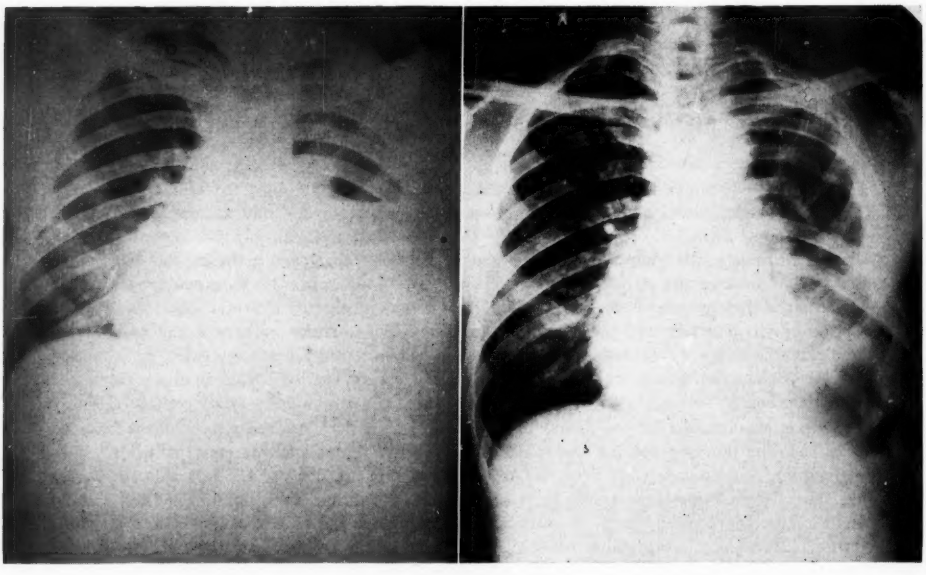
Upon examination he was found to be a poorly nourished, colored man who sat propped up in bed, continuously holding the sputum cup because the cough was most persistent. The mediastinum and trachea were displaced to the right. He did not use the left side in breathing. There were three sinus tracts in the left axillary draining very putrid-smelling, creamy exudate. As he coughed, large quantities of this offensive drainage spewed through these sinuses. Merely by compression of the chest and leaning to the left, the patient drained approximately 500 c.c. of such exudative substance.

The left lung was virtually obliterated. The left apex was filled with air and the base with findings of fluid (Fig. 8, *A*). There was a shift of the mediastinum to the right and increased peribronchial shadows in the right base. Another pertinent finding was a large,



A. *B.*

Fig. 7.—*A*, This film shows a homogeneous density over the entire left lung field. *B*, Eight days after an exploratory thoracotomy, when the pus was evacuated and decortication effected, it is noted that the lung has completely re-expanded and there is no residual fluid in the pleural cavity.



A. *B.*

Fig. 8.—*A*, Air and fluid have completely compressed the left lung and displaced the mediastinum to the right. *B*, This film, made seven weeks after operation, demonstrates a re-expanded left lung and elevated diaphragm.

protruding herniation through the right rectus incision. Viscera and peristaltic movements could be felt and seen through a thin skin that was the only protection of the abdominal cavity. The mass, however, was reducible as nothing else abnormal was detected in the abdomen.

Penicillin therapy was given intramuscularly and by inhalation. Since the hematocrit reading was only 24, the patient received four transfusions. The hematocrit reading then rose to 51 (it is interesting that the first 1,000 c.c. of blood raised the hemoglobin, red blood cells, and hematocrit appreciably little). Lung mappings did not disclose any bronchopleural communication although it was certain such existed. Bronchoscopic examination uncovered excessive, odorous exudate coming from the left main stem bronchus which appeared compressed.

Five days after entrance into the hospital a thoracotomy was performed. The left sixth rib was excised along with the draining sinuses in this region. A portion of the seventh rib posteriorly also was removed. Upon entering the pleural cavity through a very thick pleura, it was found that the diaphragm was elevated and fixed. The purulent material was all aspirated. It was estimated that some 1,800 c.c. of exudative clumps of fibrin were evacuated. The lung was tenaciously bound down by a thick, organized capsule. This was carefully and laboriously removed. Bleeding was extensive but the lung re-expanded about 85 per cent of its normal extent. It was impossible to identify a bronchial fistula, which undoubtedly was there. During the operation the patient received 2,000 c.c. of whole blood and 1,000 c.c. of normal saline solution. A No. 26 French catheter in the second anterior intercostal space ceased to bubble on the third postoperative day and was removed. The posterior one, however, was left in place for seven days.

Ten days after operation, the ventral hernia was repaired with silk under general anesthesia and the postoperative course for this was satisfactory until twelve days afterward, when he developed an abscess in the incision of the repaired abdominal hernia. This complication prolonged the hospitalization another fifteen days. Two months later (Fig. 8, *B*) the patient was making his own living, working in an office.

Comment.—This case exemplifies an individual who, after ten months of suffering from a debilitating illness and with the inconvenience of pus continuously draining from his side, was rehabilitated by decortication. It is only fair to speculate that such a case could have withstood this same procedure originally, when the treatment of the empyema was inadequate for which reason it is felt that there is a place for exploratory thoracotomy in cases that do not readily react to conservative measures. Regardless of the size of the empyema, it seems that the patient should be given the advantage of an exploration for decortication, lobectomy, or pneumonectomy as the underlying condition may warrant. In some cases, however, it is prudent to delay any extirpative surgery for a later date.

SUMMARY

1. The incidence of empyema secondary to lobar and lobular pneumonia is now less frequent than empyema secondary to heretofore uncommon etiologic factors, such as ruptured bronchiectatic abscess, infected hemothorax, ruptured subdiaphragmatic abscess, traumatic empyema, ruptured pulmonary abscess associated with or without malignancy, and as a consequence of inadequate surgery for postpneumonic empyema.

2. The advantages of an exploratory thoracotomy in cases of empyema that do not satisfactorily respond to conservative therapy are emphasized.

3. The rationale of early decortication in empyema thoracis is reaffirmed.

4. This radical attack is justified only after (a) preoperative studies for the underlying pathology have been exhaustive; (b) the hematocrit reading is 50 or above with ample blood available for the operation; (c) penicillin, 50,000 units every three hours, or streptomycin, 2 Gm. daily, has been given pre- and postoperatively; (d) a competent anesthetist trained in endotracheal anesthesia is assured, which is one of the most important factors.

5. Representative case reports are given in an attempt to establish the indications for thoracic exploration, which in reality is conservative, by reducing the morbidity and preventing economical and psychologic bankruptcy.

6. In this series of pleural empyema there have been no deaths, and all have been fully rehabilitated in a relatively short time.

CONCLUSION

After intelligent conservative measures, it is my opinion that an exploratory thoracotomy is the treatment of choice in present-day pleural empyema, otherwise we are apt to be temporizing. If one chooses not to explore these cavities one must willingly accept the likely consequences of a long morbidity superimposed on the inconvenience of a drainage tube or a sinus tract that necessitates much care. It must be borne in mind that the compressed lung may never completely re-expand unless decortication is carried out. It is agreed, however, that in some incidences this is merely treating the effect of the underlying cause. Therefore, in certain individual cases, as with these, it may be expedient to extirpate the cause while treating the effect. Such decisions, though, are a matter of surgical judgment and cannot be determined by a slide rule.

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STRANGULATION OF THE STOMACH AND TRAUMATIC DIAPHRAGMATIC HERNIA

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THE majority of patients presenting a traumatic diaphragmatic hernia are relatively free of symptoms referable either to the respiratory or gastrointestinal systems. As a rule these individuals do not have the ulcer complaints and dietary discomforts which are characteristic of congenital or hiatal hernias. Diagnosis of the traumatic type is for the most part suspected on a routine chest film following a history of left-sided trauma, and confirmation is obtained by barium study of the gastrointestinal tract. Surgical repair of the diaphragm with the replacement of the abdominal contents is strongly recommended in the traumatic cases in particular, as the results are so satisfactory, and because of the danger of strangulation of the eviscerated bowel in the untreated cases. A review of the literature reveals that strangulation in an abnormal diaphragmatic opening due to trauma does not occur commonly, but when it does develop is of sufficient gravity to justify the over-all recommendation of surgery before such a complication occurs. For this reason it is felt that our cases are of interest both in their similarity and progress.

Two cases of traumatic diaphragmatic hernia with acute strangulation of the stomach are presented. In both instances the diagnosis was made preoperatively, surgical treatment was performed transthoracically, the stomach was the only abdominal viscus involved, and successful repair was obtained. Both cases occurred in young men in the military service, and were brought about following dietary indiscretion. In neither patient had the diagnosis of hernia been made prior to the acute illness. It is our purpose briefly to present the history, physical findings, and progress in these two cases with a review of the literature and reported cases to date.

CASE 1.—This 24-year-old Mexican soldier was admitted to the 73rd Evacuation Hospital in Los Angeles on July 4, 1942, and shortly thereafter was transferred to Hoff General Hospital with the diagnosis of left traumatic diaphragmatic hernia. The history revealed that on July 3, 1942, following a long convoy ride and several glasses of beer, he became nauseated, vomited, and complained of sharp pains in the left lower chest and upper abdomen radiating to the left shoulder. This pain increased following vomiting and deep breathing. The past history was not contributory with the exception that on Oct. 14, 1940, he received a stab wound in the lower left thorax for which he was hospitalized in the Los Angeles County Hospital for about four days, following which he developed the sole complaint of considerable gas which was unaltered by food. The period of discomfort lasted nine or ten days and thereafter subsided completely. At the time of the hospitalization in 1940 no roentgen studies were taken. During the interval between the stabbing and the present illness there were occasional brief episodes of anorexia and nausea, but no vomiting. The

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Fig. 1.—Upright chest roentgenogram taken on admission showing herniated viscus with suggestion of thickening of stomach wall.

Fig. 2.—Barium swallow demonstrating complete obstruction at lower esophagus.

pertinent physical findings on July 4 were an old stab wound scar in the ninth interspace on the left side of the chest in the posterior axillary line; slight diminution of expansion of the left lower part of the chest; and impaired resonance and dullness to percussion over the anterior lower left chest with flatness in the posterior area. There was moderate tenderness to palpation in the left upper abdomen, but no evidence of rigidity or peritoneal rebound tenderness. On the day of admission, July 4, 1942, an upright roentgenogram of the chest showed what appeared to be a hollow viscus, most likely stomach, in the left pleural cavity (Figs. 1 and 2). A barium swallow taken at the same time showed complete obstruction at the lower end of the esophagus (Fig. 3). The diagnosis of complete obstruction having been established the patient was prepared for surgery. Using a posterolateral approach the left chest was opened through the bed of the eighth rib. On entering the pleural cavity a large blue-black mass was immediately encountered which was apparently hollow viscus under great tension. This extended upward into the pleural cavity for approximately 25 cm. Rib

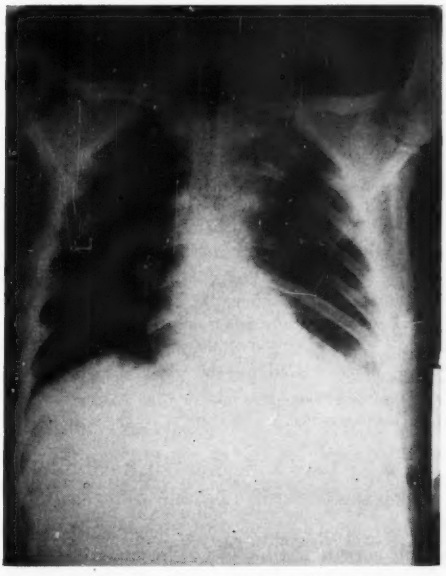

Fig. 3.—Upright chest roentgenogram taken three days following surgical repair of hernia.

spreaders were introduced and 250 c.c. of thin serosanguineous fluid having a definite musty odor was removed from the left pleural cavity. Careful exploration revealed that the mass described was strangulated stomach. No other organ was present in the thoracic cavity. Apparently the stoma of the ruptured diaphragm was relatively small and lay just mesial to the dome. Decompression of the stomach was attempted by having an assistant pass a stomach tube, and after considerable difficulty a large Ewald tube was passed beyond the hiatus into the stomach proper and aspiration of a large amount of foul thick bloody fluid and gas was effected. With the stomach collapsed it was apparent that the wall was considerably thickened and a Y-shaped incision in the diaphragm was necessary in order to return the organ to the abdominal cavity. This accomplished, the ribs were temporarily approximated and the lung re-expanded. At this time the patient's condition was very poor and a transfusion was commenced. The condition improved somewhat and the stomach was again brought into the pleural cavity. Careful examination indicated a slight change of color of the stomach wall with more of a purplish hue except in several small areas. Whether or not the organ was viable could not be determined accurately, but it was obvious that any

attempt to do a resection would undoubtedly result in a fatality on the operating table. Following a fifteen-minute application of hot packs there was a further change in the color of the stomach wall and it seemed definitely improved. The organ was replaced into the abdomen without further manipulation and the chest was rapidly closed after crushing the phrenic nerve just below the pericardium. The chest was closed in layers without drainage. A roentgenogram of the chest taken three days following surgery showed complete re-expansion of the left lung and an elevated left diaphragm. Recovery was uneventful except for a high degree of atony of the stomach which occurred immediately following surgery. This complication was followed closely by gastroscopy and roentgenograms and improved slowly to the point of complete recovery within six months when the patient was returned to full duty.

CASE 2.—This 28-year-old soldier was admitted to Fitzsimmons Hospital on Jan. 26, 1946, having been ill approximately two weeks. Eighteen months before he had received a bayonet wound of the left lower chest and was hospitalized for three weeks. Roentgen studies of the chest were taken at the time of the injury and were not available for comparison, but according to the patient no active treatment was necessary and he recovered rapidly without residual symptoms. He remained well until fourteen days prior to admission when, following the ingestion of a heavy meal, he became nauseated. The patient had no acute pain, but the nausea continued and he had intermittent vomiting attacks from then until admission to the hospital. He had been unable to take food of any kind for three days and the temperature was slightly elevated during the twenty-four hours prior to admission. On entry the patient had a temperature of 101.2° F., pulse 118, and respirations 24. He was complaining of severe epigastric pain which had commenced four hours previously and was accentuated on deep breathing. Flat films of the chest revealed herniation of the stomach, and barium studies showed complete obstruction. After fortifying the patient with intravenous fluids a left thoracotomy was performed. On entering the pleural cavity approximately 100 c.c. of clear amber fluid was encountered. The upper third of the stomach was in the pleural cavity and the fundic portion was rather densely adherent to the left lower lobe. The stomach wall was slightly hyperemic, but there was no evidence of serious interference with circulation. After enlarging the opening of the diaphragm the stomach regained its normal color promptly, was returned to the abdominal cavity, and repair was accomplished. The patient made a satisfactory recovery and was returned to full duty.

While these two cases bear a definite similarity to each other there are certain points on which there is considerable difference. Both individuals had practically the identical portions of the stomach involved, yet Case 1 was of shorter duration and represented a far more urgent surgical emergency. Case 2, as evidenced by the adhesions of the fundus to the left lower lobe, was probably slower in developing obstruction and the edema at the hiatal ring contributed to the general constriction. It is not without the realm of possibility that spontaneous reduction of the stomach might have recurred in the second case if surgery had been delayed, but such a development would have been most remarkable and certainly not to be relied upon. There is no doubt that surgery was a life-saving measure in the case of the individual first described, inasmuch as even manual reduction of the strangulation at the operating table was a difficult procedure.

Much has been written during the past several years on the general subject of diaphragmatic hernia, particularly with regard to the mechanics and physiology involved. In 1934 Hedblom[1] reported 1,408 cases of diaphragmatic hernia, mostly collected from the literature, of which 821 were congenital and 587 traumatic in type. In reviewing the literature on the subject of acute strangula-

tion of the stomach, one is impressed with the few reports concerning this complication. This is evidenced by the fact that there have been but four cases reported in which the stomach has been the strangulated organ. The first case report[2] concerned a 25-year-old psychotic man who stabbed himself in the left side of the chest in 1920. Nothing remarkable occurred then or for the following few years other than a lack of appetite, which was felt due to his psychotic nature. In 1924, four years following the stab wound, the patient first showed signs of complete obstruction as evidenced by vomiting, fever, and general malaise. After a short period of observation the diagnosis of a strangulated stomach and a diaphragmatic hernia was made and a laparotomy was performed. An enormously dilated stomach was found herniated through a small opening in the left diaphragm, and trocar aspiration of the stomach was performed before it could be returned to the abdominal cavity. The patient did poorly immediately following surgery and died on the first postoperative day.

The second case showing definite involvement of the stomach in a hernia of this nature was reported by Wright.[3] This patient was a 52-year-old woman admitted to the hospital with acute epigastric pain of sudden onset four days previously. The history revealed that she had suffered a severe blow to the upper abdominal region nine months before, but had experienced no difficulty since that time. On admission to the hospital she complained of severe pain in the left shoulder, and in order to breathe she had to sit up and lean forward. The diagnosis of intestinal obstruction was made and a laparotomy disclosed the stomach, spleen, and omentum incarcerated in a left diaphragmatic hernia. Reduction was accomplished, and the stomach was purplish, but appeared viable. The hernia was repaired and the patient died two hours following surgery.

The third case, appearing in the Spanish literature,[4] was reviewed by DeBakey. Here the author reported a strangulated diaphragmatic hernia in which the stomach was the only organ involved. Diagnosis was made preoperatively from roentgenograms showing a gaseous circle with a liquid level above the left half of the diaphragm. The onset was sudden two days before admission followed by a few hours of sudden continuous pain in the upper left abdomen and at the base of the left thorax with considerable respiratory distress. Three years before admission the patient had had a repair of the left strangulated diaphragmatic hernia. Left thoracotomy was performed on both occasions and the stomach containing diaphragmatic hernia was reduced with excellent results. This is the only case on record in which both strangulated hernias were successfully treated.

The fourth case report[5] described a 37-year-old man who was admitted to the hospital complaining of severe abdominal pain. Physical findings revealed a left pleural effusion which needle aspiration proved to be bloody in character. Under supportive treatment the patient died in forty-eight hours, and post-mortem examination revealed a left diaphragmatic hernia with strangulation of the stomach and omentum and a large quantity of bloody fluid in the left pleural space. The stomach was gangrenous throughout the upper third. No history of previous chest or upper abdominal injury was

given, but from the description of the diaphragmatic opening the supposition that the opening was traumatic in nature is probably justified. The authors quoted a statement by Knaggs made in 1904 with regard to the mechanism or physiology concerned in strangulation of the stomach which seems worthy of repetition even more than forty years later: "The ulceration that occurs in strangulation of the stomach seems to be different from ulceration and strangulation of the intestine. In the latter it begins in the mucous membrane surface and extends to the outer coats. In the case of the stomach the available evidence points to extreme distension, producing splits through the integrity of the sodden and rotten mucous membrane. Further ulceration does not appear to have been noted at the seat of constriction, the most common place for it in the intestine."

In considering the four cases reported, there are several factors which seem significant. There are doubtless other cases of this general nature which are not listed in the available records. Mortality for this series is 75 per cent. Three patients were treated by surgery and one was given supportive therapy. The one not operated upon died within two days after coming under observation and showed a gangrenous stomach at post-mortem examination. The sole surviving patient[4] was most unusual in that a strangulated diaphragmatic hernia was repaired on one occasion only to be followed by a recurrence of the exact mechanism three years later. In both instances successful repair was accomplished through a transthoracic approach. It is not so stated, but it is doubtful that serious interference in blood supply of the stomach was present on either occasion. The only patient operated upon who showed definite evidence of circulatory disturbance at surgery died. This patient was actually ill for four days and was operated on by the abdominal approach. In the third fatal case[2] the patient had been acutely ill for ten days prior to laparotomy, which may have contributed to the shock of surgery and fatal result.

It is not the purpose of this paper to cite two isolated cases of traumatic diaphragmatic hernia with acute strangulation of the stomach as therapeutic triumphs. The operation in itself is not unusual nor technically difficult and the importance of considering the subject lies in the recognition of the defect and its surgical correction before strangulation occurs. Once the diagnosis of a traumatic diaphragmatic hernia has been made, surgical repair should be performed even though the patient is entirely free of symptoms. In the quiescent type, reduction of the hernia at operation is usually a simple procedure and the repair of the diaphragmatic opening relatively easy. The statistical evidence of acute strangulation in hernia in this location is extremely difficult to estimate, largely due to the fact that undoubtedly many cases of this type are never diagnosed, probably on account of their lack of symptomatology, and hence are never observed. This would further indicate that it is only the occasional case which goes on to strangulation. In spite of the obviously small percentage of cases which eventually require emergency surgery for a strangulated abdominal viscus, even such a rare occurrence justifies routine repair of all such abnormalities as soon as the diagnosis is established.

SUMMARY AND CONCLUSIONS

1. Two cases of acute strangulation of the stomach and traumatic diaphragmatic hernia occurring in the military service are presented. Successful results were accomplished by prompt transthoracic reduction and repair of the defects.

2. A review of the available literature on the subject is given. Four similar cases have been reported with the mortality of 75 per cent.

3. All traumatic diaphragmatic hernias should be repaired as soon as the diagnosis is made and certainly before signs of obstruction occur.

4. The transthoracic approach is the method of choice in the repair of hernias of this type.

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TRAUMATIC CHYLOTHORAX

A Review of the Literature and Report of a Case Treated by Mediastinal Ligation of the Thoracic Duct

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INJURY to the thoracic duct in the chest and the ensuing escape of chyle into the free pleural space constitutes a grave catastrophe in which there is scarcely an even chance of recovery. The seriousness, the rarity, and the fascination of this condition have led to the sporadic reporting of cases by many observers with diverse opinions and speculations concerning the management of the problem. Most of the therapeutic measures have followed the hope that spontaneous closure would occur and have in general been supportive in character. Ligation of the duct in the chest has been mentioned as physiologically feasible but has not been successfully carried out as a part of therapy. The purpose of this paper is to present a case of traumatic chylothorax with recovery following transpleural mediastinal ligation of the thoracic duct.

The thoracic duct is a thin tenuous structure which courses from the cisterna chyli upward along the vertebrae in front of the right intercostal vessels and behind the esophagus to the upper mediastinum where it diverges to the left to pass behind and above the great arteries in the neck to empty into the subclavian vein at its junction with the left jugular vein. Lee,[1] with meticulous care, has demonstrated that there are many small collateral branches which are in conjunction with the main duct and that some of these communicate by small filaments with the lumbar, intercostal, and azygos veins. This anatomic observation has been further substantiated by the experience of surgeons[2-5, 10] who have ligated the thoracic duct in the neck with impunity and by investigators[1, 6, 7] who have occluded it in animals without deleterious effect. The thoracic duct and its tributaries serve as the channel through which chyle is carried from the digestive system to the venous circulation. Histologic study of the duct shows that in its wall there are fibers of smooth muscle[8] and it has been indicated that rhythmic contractions of these fibers help to propel the chyle upward along the duct. The changes in the intrathoracic pressure with respiration and the accumulation of chyle in the cisterna chyli are also most important factors in its flow. Under normal conditions the pressure in the duct is very small but it has been observed that after ligation it can reach 35 cm.[9] of water. The rate of flow and the volume carried are dependent, within a certain range, upon the amount and character of the oral intake of the individual. It has been estimated that the flow of chyle in man varies between .38 and 3.9 c.c.[10] per minute or 60 to 190 c.c.[4] per hour, or approximately 2 liters a day.

From the Surgical Service and the Chest Group of the Hartford Hospital.
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Chylothorax is by definition the condition which results when chyle accumulates in the free pleural space. This occurs in the presence of serious and frequently fatal disease such as venous obstruction to the superior vena cava, lymphatic block in filariasis, erosion of the duct or its radicals by malignant neoplasm, or tuberculosis.[11] The escape of chyle from trauma, which may be direct or indirect, constitutes the main theme of this paper.

When chylothorax develops following direct laceration which severs the duct as in bullet wounds,[4, 14, 28, 31] surgical accidents[16, 23, 27, 29] and fractures of adjacent vertebrae,[20] or following indirect injury which tears it by concussion, or traction from external blows[15, 18, 30] or even coughing, there is always a latent period before symptoms appear. This interval is usually three to seven days but may be as long as months.[18, 25] The delay is the time required for the chyle to escape from the extrapleural mediastinum into the pleural space and fill it to the point where the volume of fluid embarrasses respiration and causes distress. In some instances, this alteration of respiratory physiology will precipitate cardiovascular collapse and shock which will respond to thoracentesis.[14, 29] In patients in whom the condition has been of long duration, accompanied by frequent thoracenteses, hypoproteinemia, weight loss, and inanition are marked due to failure of essential products of nutrition to enter the circulation.

The diagnosis of chylous effusion is established by the character of the pleural fluid. Chyle is a milky, thin fluid with a specific gravity of 1.010 to 1.021, and alkaline in reaction. It contains lipoid substances which are stained by sudan III. The values for electrolytes, protein, sugar, and cholesterol in chyle approximate those of the serum but are subject to wider variations which may be a direct reflection of the diet.[10] Chyle is a vehicle for the mobilization of proteins which, in the presence of hemorrhage, are redistributed and made available for essential functions.[6] Chyle is bacteriostatic as demonstrated by the low incidence of empyema and by the many sterile cultures after repeated thoracenteses in the reported cases. This has been substantiated in the Hall-Wilson Laboratory of the Hartford Hospital where cultures of *Eschericha coli* and *Staphylococcus aureus* failed to multiply in pure chyle. Table I indicates the results of these observations. Table II records the values for some of the constituents of chyle as well as the corresponding blood levels. It also indicates the date and amount of the various chest taps.

Traumatic chylothorax is an extremely unusual disease, and there are only sixty-nine cases of it recorded in the accessible literature. Shackelford and Fisher[12] in 1938 reported two cases and presented in tabular form a very excellent summary of thirty-nine other cases previously recorded in the literature. Eleven incomplete reports were discarded due to the lack of detailed description. Since their paper, seventeen additional cases have been reported, and they are summarized in Table III, making a grand total of sixty-nine. The therapeutic results for the series of fifty-eight cases which could be analyzed show a gross mortality rate of about 45 per cent and are given in Table IV. The ages ranged from 9 to 62 years; forty-seven were males, and eleven females. This is to be expected in a traumatic disease.

TABLE I. BACTERIOLOGIC STUDIES WITH CHYLOUS FLUID*

PERCENTAGE OF CHYLE	PERCENTAGE OF PHENOL RED BROTH	REACTION IN 24 HOURS	GAS IN 24 HOURS	PROTEIN CHANGES
		E. Coli W.		
100	0	Alkaline	0	0
50	50	Acid	Plus	Coagulation
30	70	Acid	Plus	Coagulation and digestion
20	80	Acid	Plus	Coagulation and 3+ digestion
0	100	Acid	Plus	Complete digestion
		Staph. 204		
100	0	Alkaline	0	0
50	50	Acid	Plus	Coagulation
30	70	Acid	Plus	Coagulation and digestion
20	80	Acid	Plus	Coagulation and digestion
0	100	Acid	Plus	Complete digestion

*Phenol red broth was diluted with varying amounts of chyle and then used as a culture medium for *E. Coli W.* and Staph. 204. The amount of growth was gauged by change in pH and volume of gas produced. In addition, change in protein content was noted.

Transplants from pure chyle to blood agar plates and endo showed 4 plus growth indicating that pure chyle is bacteriostatic and not bactericidal.

TABLE II. VALUES OF IMPORTANT CONSTITUENTS IN CHYLE AND CORRESPONDING BLOOD LEVELS

DATE 1946	AMOUNT (C.C.) CHYLE	COLOR CHYLE	N-P-N (MG. %) CHYLE	N-P-N (MG. %) BLOOD	PROTEIN (GM. %) CHYLE	PROTEIN (GM. %) SERUM	SUGAR (MG. %) CHYLE	SUGAR (MG. %) BLOOD	CHOLESTEROL (MG. %) CHYLE	CHOLESTEROL (MG. %) BLOOD	CALCIUM (MG. %) CHYLE	CALCIUM (MG. %) BLOOD	PHOSPHATE (MG. %) CHYLE	PHOSPHATE (MG. %) BLOOD	CHLORIDE (MG. %) CHYLE	SP. GR. CHYLE	PH CHYLE
2/5	300	Thin, milky															
2/12	1700	Chylous	25	26	3.8	5.8	122	80	97	120	7.5		4.6		660		
2/20	700	Thin, milky														1.017	7.7
2/21	900	Thin, milky			3.7	6.5		93	91			11.5		3.4		1.021	
2/24	1050				3.1		119		94							1.010	
2/28	2100	White, opaque	21		3.0		122		92		7.8		4.1		735		

TABLE III. SUMMARY OF CASES OF CHYLOTHORAX REPORTED IN THE LITERATURE SINCE 1930[12]

AUTHOR	DATE	AGE AND SEX	NATURE OF TRAUMA	CLINICAL COURSE	SIDE	THERAPY	RESULT	AUTOPSY FINDINGS
1. Strauss[14]	1936	24 F	Bullet wound of chest	Ten days later shock and dyspnea, diagnosis chylothorax made by thoracentesis, recovered after 3 mo.	R	Repeated thoracenteses and pneumothorax. 36 aspirations—79,800 c.c. removed	R	
2. Brown[15]	1937	? F	Auto accident with collision	Two months later chylous ascites; laparotomy done, seventeen days P.O. dyspnea with bilateral chylothorax, died 4 mo. after injury	R & L & A	Laparotomy. Repeated thoracenteses. Posterior drainage of mediastinum	D	Interruption in continuity of thoracic duct 2.5 cm. above the diaphragm. Fibrosis about the distal end occluding the lumen. Chylous fluid in both pleural cavities and in the peritoneal cavity
3. Douday, Dupuy, and Bouvrain[16]	1938	30 M	Lysis of adhesions of left lung by thoracoscope for Tbc.	The next day fever and dyspnea appeared and fluid above the diaphragm; tap showed it to be milky	L	Seven days later 100 c.c. of milky fluid removed. Fifteen days later complete recovery from chylothorax	R	
4. Fujita, Bando, and Sugishita[17]	1939	10 M	Struck at play in left infra-clavicular region	Fracture of first left rib and chylothorax developed on left	L	Repeated chest taps. Incision made in left neck and cervical thoracic duct compressed with a pack. Recovered after three months	R	
5. Gordon[18, 19]	1940	31 M	Severe blow on chest while at work	One month later cough, L.M.D. tapped chest and found milky fluid, cough persisted, bed rest, 7 mo. later went to hospital, discharged after 4 mo. requiring aspiration once a month	R	Repeated thoracenteses. 950 to 1500 c.c. removed on frequent occasions for three months. Chest irrigations with azochloramid. Fluid diminished	R	
6. Cellan-Jones, and Murphy[20]	1940	32 M	Severe blow and crush of chest and mid-back while at work	Three days later developed dyspnea and collapse, downhill course; died 15 days after injury	R	Repeated thoracenteses. Twenty-seven pints removed in twelve days	D	At post-mortem ninth and tenth vertebrae were fractured and the thoracic duct torn as well as the parietal pleura communicating with the right chest cavity.

R, Right
L, Left

A, Abdomen
R, Recovered
D, Died

(Table III continued on following pages.)

TABLE III—CONT'D

AUTHOR	DATE	AGE AND SEX	NATURE OF TRAUMA	CLINICAL COURSE	SIDE	THERAPY	RESULT	AUTOPSY FINDINGS
7. Matson and Stacy[21]	1940	21 M	Gunshot wound of right chest	Patient in shock, right chest showed pneumohemothorax, tapped repeatedly, on twelfth day chyle first noted	R	Repeated thoracenteses. 22,440 c.c. Gomenol and oil injected. By end of five months patient well.	R	
8. Hoyer[22]	1941	56 M	Hit by auto	Fractures of the ninth, tenth, and eleventh ribs on right; 5 days later chylothorax detected	R	Repeated thoracenteses. 6,600 c.c. total. Discharged well forty days after accident	R	
9. Smith and Woliver[23]	1941	17 F	Surgical accident	Thoracic duct injured and not ligated during resection of a Ewings tumor of the tenth rib and vertebra, third P.O. day dyspnea developed, well fifteenth P.O. day	R	Thoracentesis on fifth P.O. day 900 c.c. In next twelve days 7,800 c.c. removed. 3,000 c.c. given intravenously. Spontaneous closure on fifteenth P.O. day.	R	
10. Dorsey and Morris[24]	1942	60 M	Fell down stairs in alcoholic stupor; fractured tenth rib	Twenty-eight days later developed dyspnea; right thoracentesis, 3,100 c.c. of milky fluid, died 46 days after injury	R	Repeated thoracenteses. 18,400 c.c. removed in eighteen days. 3,500 c.c. given intravenously. Died of inanition.	D	The thoracic duct was injected with India ink and showed a small puncture-like opening in the duct lying over the 12th thoracic vertebra. 9th and 10th ribs fractured. No fracture of the vertebra
11. Little, Harrison, and Blalock[25, 26]	1942	17 F	Auto accident	Seven weeks later dyspnea; 1,500 c.c. chyle from left chest, later right chest and peritonium involved	R & L & A	Repeated taps of chest and abdomen. Intravenous reintroduction of total of 11,100 c.c. of chyle. After eighteen months abdominal chyle still accumulated. Over 500 liters were removed from chest and abdomen. Patient finally died.	D	Patient died outside of hospital. Autopsy not obtained
12. Whitcomb and Scoville[27]	1942	39 F	Surgical accident	Thoracic duct injured and recognized and clipped during right sympathectomy; fourteenth day severe dyspnea, died in anaphylactic shock	R	Repeated thoracenteses. Intravenous chyle was given slowly. After 75 c.c. patient went into anaphylactic shock and died.	D	Permission refused.

13. Schnug and Ransohoff[28]	1943	23 F	Bullet wound between fifth and sixth ribs	Immediate exploratory laparotomy, 6 days later developed dyspnea, right chest tapped and chyle found; 56,450 c.c. removed by tapping over 33 day period	R	Right phrenic crushing on 15th day. 16,800 c.c. chyle given by vein. 32nd day 50 c.c. of sterile broth injected into chest to stimulate fibrinous closing of tear. Chyle stopped 33rd day.	R	
14. Peet and Campbell[29]	1943	47 M	Surgical accident	Bilateral splanchnicectomy for hypertension, 11 days later chylothorax with sudden cardiovascular collapse and dyspnea	R	Thoracentesis three times in five days. 10,700 c.c. removed. Chyle given intravenously and intrasternally. Ligation of thoracic duct attempted. Patient died suddenly.	D	Post mortem revealed the thoracic duct to be completely interrupted at the level of the eleventh thoracic vertebra. No satisfactory explanation for sudden death was found
15. Florer and Ochsner[30]	1945	14 M	Fell from iron framework	Four days later dyspnea, tap 1,100 c.c.; no fractured ribs or vertebrae found	R	Sixteen taps. 26,000 c.c. removed. Right phrenic crushed. Intravenous chyle given. Fat free diet. Died twenty-four days after injury.	D	Perforated duodenal ulcer. The thoracic duct showed marked narrowing 3 cm. above cysterna chyle. There was a small rent in the pleura at the eleventh intercostal space. No fractures found.
16. Loe[4]	1946	? M	Bullet wound in neck in action on Saipan	Arrived on hospital ship in shock and respiratory distress	L	Taps twice daily to relieve dyspnea. Total of 18,000 c.c. removed in nine days. Fourth day recognized as chyle. Ninth day duct ligated in neck	R	
17. Berry[31]	1947	28 M	Shrapnel wound in left chest in action in Germany	Arrived army hospital in fair condition with respiratory distress; x-ray showed large foreign body in left chest, thoracotomy done, fragment removed from upper posterior chest wall, 48 hr. later developed dyspnea, thoracentesis done; 800 c.c. blood-stained chyle obtained	L	Repeated thoracenteses daily for eight days. Spontaneous cure by fourteenth day. 10,375 c.c. removed in all	R	

TABLE IV. SUMMARY OF THERAPEUTIC RESULTS IN FIFTY-EIGHT CASES ANALYZED IN THE LITERATURE

	RECOVERED	DIED	UNKNOWN
Total of fifty-eight cases	31 (53%)	26 (45%)	1 (2%)
Nonoperative therapy	22	19	1
Rib resection	6	4	
Cervical ligation	2		
Phrenic crushing	1	1	
Posterior drainage		1	
Posterior ligation		1	

The most striking feature in the management of these patients is the multitude of ingenious suggestions which have been considered or executed in the treatment. Frequent thoracenteses for the relief of respiratory distress have been almost universally used. Pneumothorax for the purpose of discouraging the flow of chyle has been done without great success. Instillation of sterile broth[28] and irrigations with azochloramid[18] and other substances[21] to stimulate fibrinous reaction have been resorted to with some benefit. The phrenic nerve was crushed twice with doubtful success.[28, 30] The intravenous[13] or intrasternal[29] injection of chyle has been done with benefit, but in one instance in the Hartford Hospital it resulted in sudden death from anaphylactic shock.[27] The intravenous administration of chyle is not safe even though it has been accomplished successfully. Restricted diets low in fluids, fat, and protein have had beneficial results in reducing the production and flow of chyle. Rib resections[12] have been done to establish an external chylous fistula, and also under the mistaken diagnosis of empyema. Surgical drainage of the mediastinum through the posterior approach has been attempted[15] with the thought that a free external flow of inspissated chyle would encourage healing of the rupture in the duct. These methods have been instituted in the hope that spontaneous closure of the mediastinal sinus would occur. Direct injury to the thoracic duct in the neck has resulted in external chylous fistula[10] or in chylothorax.[4, 17] Both of these conditions have been controlled by ligation of the cervical thoracic duct. Retropleural ligation of the thoracic duct for the control of chylothorax resulting from a surgical accident during a bilateral splanchnicectomy has been done without success.[29]

When conservative measures fail, and it becomes clear that spontaneous repair and closure are unlikely, then one is justified in risking a direct attack through the chest at or below the site of the rupture with the intent to ligate the duct low in the mediastinum. The case which is presented in this paper establishes the fact that low ligation of the thoracic duct is a feasible procedure. The chylothorax promptly ceased and the patient has remained free from it for over one year.

CASE REPORT

Mrs. S. S. (Hartford Hospital, No. 530-622), a 35-year-old married machine operator at a soap factory, came to the outpatient department on Jan. 22, 1946, with a chief complaint of shortness of breath. Two months before, while visiting in Maine, she developed a respiratory infection which was thought to have been a mild pneumonia and which was accompanied by very severe paroxysms of coughing productive of a moderate amount of sputum. The patient was confined to bed for one week under medical care but was not hospitalized. Following this acute episode she was never entirely well. A chronic, nonproductive cough

persisted, and shortness of breath was experienced when walking or climbing stairs. About three weeks before entry to the outpatient department these symptoms quite suddenly became markedly worse, and the patient could not walk because of severe dyspnea from the slightest exertion. She was referred by her doctor to the clinic. During the recent illness she lost ten pounds in weight. Previous general health had been good. The family history was significant in that her mother had died at the age of 29 years of tuberculosis, and her father had died of an unknown cause.

The initial physical examination disclosed the following positive findings: height 5 feet 6 inches, weight 112, temperature 97.8° F., pulse 102, respiration 22, and blood pressure 108/70. Her appearance was that of a thin, white woman in no acute distress and with good color. The chest showed good expansion but very little excursion of the left diaphragm. The pulmonary fields were resonant everywhere except on the left in the lower portion posteriorly and in the axillary line. Breath sounds were diminished in this area. There was an area of egophony immediately below and medial to the left scapula along the spine. No râles or rhonchi were heard.

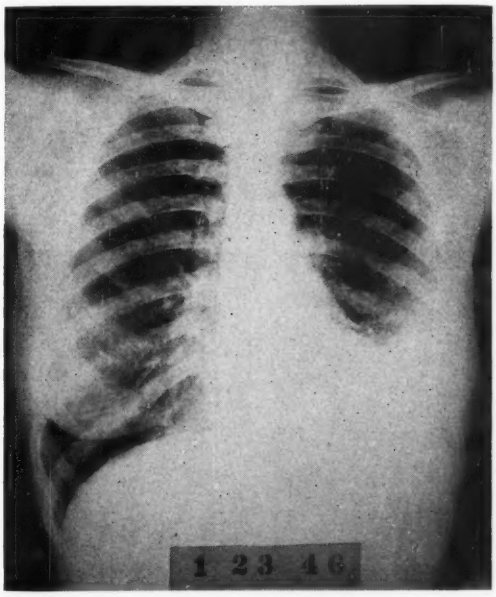

Fig. 1.—Initial chest x-ray view taken in the outpatient department on Jan. 23, 1946, showing fluid at the left base.

The initial laboratory findings were as follows. Urine: acid, specific gravity 1.020, albumin 1 plus, sugar 0, casts 0, cells 0. Blood: hemoglobin 91 per cent, hematocrit 51, basal sedimentation rate 15 mm. per 1 hour, polymorphonuclears 70 per cent, lymph 30 per cent, red blood cells and platelets appeared normal. Serology: Mazzini and Hinton negative. Electrocardiogram was done and was not diagnostic of cardiac abnormalities. X-ray examination of the chest was reported as follows: Films of the thorax showed a moderate amount of pleural fluid or thickening at the left base, the cause for which was not apparent. There was generalized exaggeration of the pulmonary structures throughout both lung fields, and an underlying pulmonary pathologic process could not be excluded from the left base where the pulmonary parenchyma was obscured by fluid.

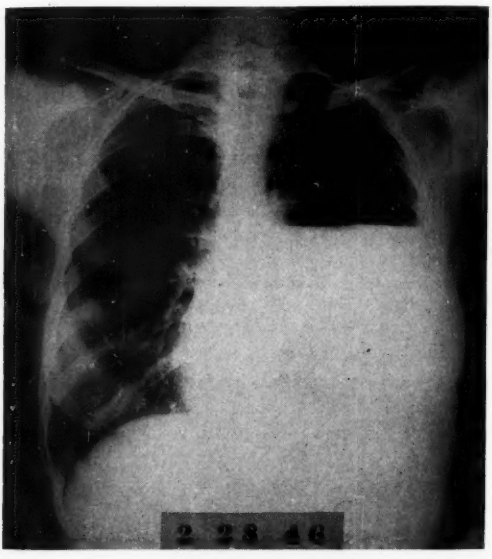

Fig. 2.—Chest roentgenogram taken just before operation showing large collection of chyle in the left chest and pneumothorax.

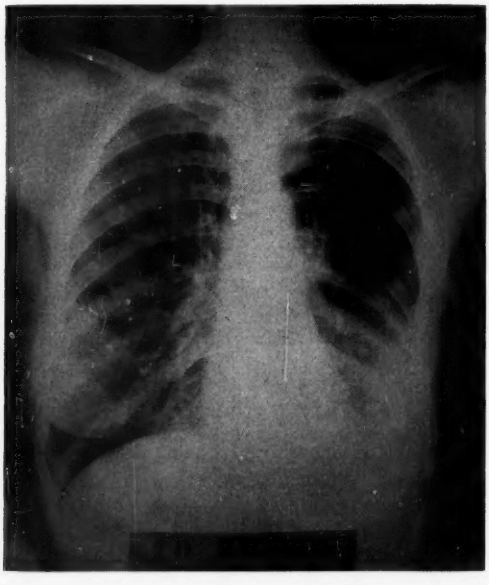

Fig. 3.—Follow-up chest x-ray picture taken eight months after operation showing some residual pleural thickening at the left base.

Two weeks later the patient made a second visit to the outpatient department and a diagnostic thoracentesis was done in the seventh interspace in the left midaxillary line, and 300 c.c. of thin, milky fluid were aspirated. A tentative diagnosis of chylothorax was made and the patient was advised to enter the hospital.

On February 9 the patient entered the hospital for observation, treatment, and study to determine the cause and course of the chylothorax. During the ensuing three weeks five chest taps were done and these indicated that the rate of reaccumulation was increasing. Pneumothorax was instituted in order to give a better radiographic diagnosis of the underlying lung and mediastinum, and in the hope of discouraging the flow of chyle. Neither objective was accomplished. Examination of the chest by fluoroscopy disclosed marked depression of the diaphragm which showed parodoxical motion and gave the impression that the diaphragm was paralyzed. Culture of the chylous pleural fluid produced no bacterial growth and inoculation of chyle with organisms showed that it was bacteriostatic. Repeated sputum examinations failed to show tubercle bacilli. The patient was placed on a low fluid, protein and fat intake which had no noticeable effect in discouraging the reaccumulation of chyle. Intravenous administration of chyle was considered and thought to be inadvisable. Inasmuch as spontaneous improvement failed to occur, it was decided that exploratory thoracotomy with ligation of the thoracic duct below the point of rupture should be attempted. Operation was done on March 1, 1946, and is described as follows.

Fig. 4.—Chyle, 2,000 c.c., removed in one tap the day before operation.

Under endotracheal cyclopropane and ether anesthesia, with the patient lying on the right side, an incision was made over the sixth rib from the anterior axillary line to the posterior end of the rib. The scapula was freed and retracted upward and a subperiosteal resection of the sixth rib was done. The fifth and seventh ribs were divided posteriorly. The pleura was opened and the lung was found to be free except for some thin adhesions posteriorly. The chest cavity contained chylous fluid which was removed with suction. The adhesions of the lung to the posterior chest wall were carefully separated by digital dissection, and the entire left lung was freed. Careful examination along the gutter in front of the aorta finally disclosed a small point adjacent and posterior to the inferior pulmonary ligament

just anterior to the aorta through which chyle could be seen to appear. The gutter anterior to the aorta was blocked above and below this point with gauze, and it was accurately determined that the chyle originated from this point and did not flow to it from a leak more distantly located. Digital and visual examination of the mediastinum above the heart showed no abnormalities. The phrenic nerve was visualized and there was no interference with it within the chest. The diaphragm also moved with respirations, indicating that there was no paralysis of the phrenic nerve as had been thought by the preoperative fluoroscopic x-ray reports. A four and one-half inch incision was made in the mediastinal pleura just anterior

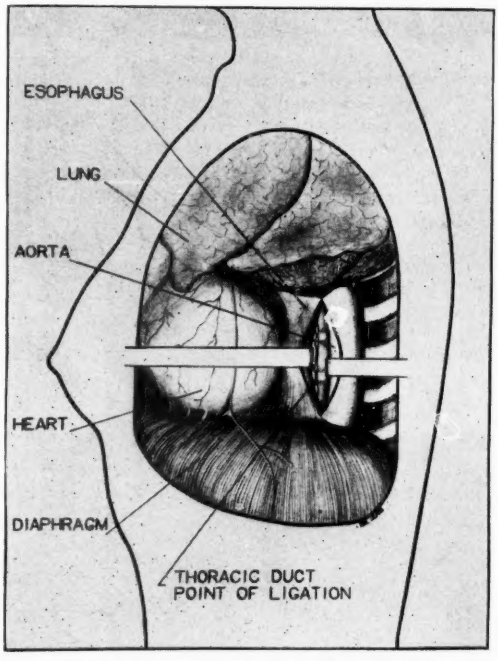

Fig. 5.—Diagrammatic sketch of the operative approach for mediastinal ligation of the thoracic duct.

to the aorta and below the inferior pulmonary ligament approximately at the level of the ninth dorsal vertebra. The esophagus was identified and dissected anteriorly. The left vagus nerve was visualized. The aorta was retracted to the left and posteriorly. Directly in front of the vertebral column behind the aorta there was identified a distinct structure the size, consistency, and color of the thoracic duct traveling anterior to the spinal column in the vertical direction. Five grains of methylene blue dissolved in water were injected by the anesthetist through the stomach tube to see if it would appear in the thoracic duct. It did not appear. Two silk ligatures were placed around the thoracic duct and tied. The duct was not divided. The incision in the mediastinal pleura was closed with interrupted silk sutures. A small amount of fibrin foam was inserted to control whatever small amount of bleeding was present. The chest wall was closed in layers without drainage. Following closure, aspiration was done anteriorly with a large needle and about 2,000 c.c. of air were removed. Penicillin, 100,000 units, was inserted through the needle. At the end of the operation the patient was in very good condition.

The immediate postoperative course was uneventful. Chest tap was done on the first postoperative day and 900 c.c. of serosanguineous fluid were withdrawn, which had no suggestion of chyle. Subsequent postoperative taps were done and were entirely free of chyle. The convalescence was slightly complicated by the development of atelectasis of the left lower lobe and the appearance of follicular tonsillitis on the twelfth postoperative day. The patient was discharged from the hospital markedly improved on April 6, five weeks following the operation. At the present time over one year has elapsed and there has been no reaccumulation of the chyle. The patient, however, has had great difficulty in maintaining her weight and there has been some residual fixation of the left chest.

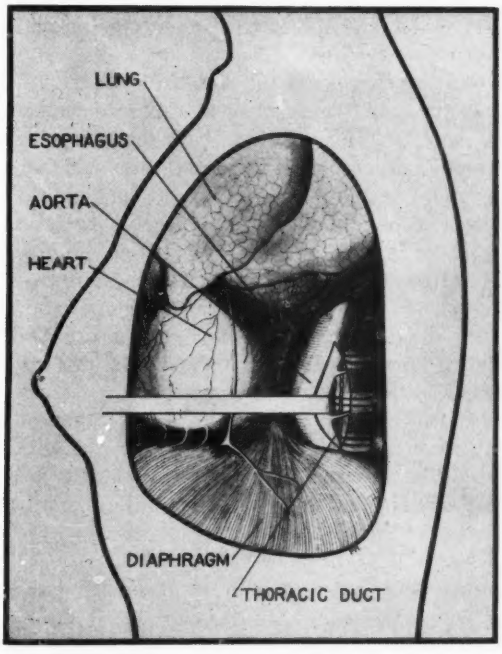

Fig. 6.—Approach used by Meade, with retraction of the aorta forward and to the right.

COMMENT

Since the preparation of this paper, Meade has successfully treated a patient with chylothorax by mediastinal ligation of the thoracic duct through the left chest. In his case the operative approach was made by incising the mediastinal pleura over the aorta and retracting the aorta forward and to the right, exposing the duct between the intercostal arteries and running along the anterior surface of the vertebrae. This approach, according to Meade, is easier than the approach through the left chest by going to the right and in front of the aorta. It is through his kindness that this alternative procedure is illustrated in Fig. 6 so that others who may undertake this operation will be advised that the thoracic duct can be reached through the left chest from the right or the left

side of the aorta. It is not intended that mention of Meade's case should be considered as a report and for this reason it has not been included in the figures of the cases reported in the literature.[32] When the chylothorax occurs in the right chest the approach should be made through a right exploratory thoracotomy and the thoracic duct identified and ligated.[33]

DISCUSSION

The diagnosis of chylothorax in this patient is evident; its etiology is obscure. The history of very severe coughing and the lack of a gross pathologic process in the chest at the time of operation forces the conception that the break in the thoracic duct resulted from the strenuous action of the diaphragm in coughing, which must be considered the traumatic agent. Chylothorax following indirect trauma probably results from the partial rupture of the thoracic duct or one of its radicals. This may not divert the entire chylous flow into the chest. The sequence of events in this case indicates that there must have been an incomplete severance of the duct. The small amounts of chyle in the chest in the initial x-ray, the long latent period, and the slow accumulation of chyle before admission to the hospital all substantiate this impression. Even if one postulates a small perforation of the main duct or the rupture of a small radical, the surgical attack should still be directed at ligation of the main duct rather than at repair of the site of injury, which would be hard to find and even harder to close. It is evident from previous reports and from this case that the chyle is not denied entrance into the blood stream, and that it must make its way through collateral channels to the venous circulation. Even though it has been demonstrated that low ligation of the thoracic duct is feasible, it is a rather difficult procedure and should be reserved for the patient who has demonstrated by the clinical course that spontaneous closure in all probability will not occur.

SUMMARY

1. The literature covering reports of traumatic chylothorax has been reviewed.

2. A case of chylothorax following the trauma of very severe coughing has been presented.

3. Mediastinal ligation of the thoracic duct for chylothorax has been carried out with cure of the chylothorax.

Acknowledgment and appreciation are extended to Dr. John T. Beebe who made the diagnosis, to Dr. Welles A. Standish who assisted at the operation and in the postoperative care, and to Dr. Ralph E. Kendall who directed the laboratory studies.

ADDENDUM

During the second year following the ligation of the thoracic duct, this patient developed severe asthma, and on June 1, 1948, died in the Hartford Hospital of status asthmaticus. Permission for an autopsy was refused. At no time during the two years after operation was there any reaccumulation of chyle in the chest, ascites, or peripheral edema. The patient had always been very thin and remained so. The usual important blood chemistry values on the final hospital admission were essentially normal. The complete picture in this case remains unknown, but the control of the chylothorax was permanent, and the author feels that the patient died of unrecognized pathology and not as a result of the ligation of the thoracic duct.

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COMBINED NITROUS OXIDE, PENTOTHAL SODIUM ANESTHESIA IN THORACIC SURGERY

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WE ARE reporting our experience with this method in 363 consecutive operations at the Detroit Tuberculosis Sanatorium between Jan. 9, 1945, and April 12, 1947. For the most part, these were poor-risk patients for surgical procedures. Two hundred thirty-two operations were on patients with advanced tuberculosis, on whom simpler methods of treatment had failed; 62 others had long-standing infections with accompanying toxemia and possible damage to liver, heart, and kidneys; 30 were carcinomas of the lung, 3 of whom were cyanotic before reaching the operating room. The youngest was 15 years, and the oldest 61 years.

Randolph and Kober[1] had reported favorably on the use of Pentothal sodium in thoracic surgery, chiefly for thoracoplasties. Only 1.7 per cent of their cases showed pulmonary complications.

Paquet[2] also reported very favorably on the use of pentothal for thoracoplasty in a series of 175 operations. He pointed out that with depressed respirations, paradoxical breathing was decreased and that to-and-fro movements of the mediastinum were easily controlled. Hemorrhage was less since pentothal did not cause peripheral vasodilatation. Orton[3] recently reported his use of pentothal for extrapleural thoracic surgery. Moyer and McKittrick[4] found that animals with open pneumothorax anesthetized with pentothal might become permanently apneic following the cessation of painful stimuli. This they attributed to either anoxia or hypercarbia depressing the respiratory centers during the experiment. We have not experienced any similar effect in our series. Heard[5] stated that the only real contraindication to pentothal is the anesthetist's inability to maintain an adequate airway.

Organe and Broad,[6] Ritter,[7] and Shane[8] had recommended the use of nitrous oxide and oxygen with pentothal and stated that this combination greatly reduced the amount of pentothal necessary to maintain anesthesia.

We began our use of this combination on extrapleural cases, and found the results so satisfactory that we quickly extended its use to all cases. Whenever the pleura was to be opened and if there were any question of maintaining an adequate airway the patient was intubated.

Maier[9] has stressed the importance of the closest cooperation between surgeon and anesthetist in thoracic surgery. Not only should the anesthetist maintain a suitable depth of anesthesia, but he must provide an adequate respiratory exchange with a minimum of effort on the part of the patient, even though the mechanism of respiration is materially altered by removal of ribs or the opening

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of the thoracic cavity. Many patients will have a lowered vital capacity from pulmonary disease, or such procedures as pneumothorax or phrenic paralysis on the contralateral side. This fact makes it desirable to use an increased oxygen tension during the course of the operation. It is imperative to maintain adequate oxygenation at all times. One must also watch for any evidence of increased CO_2 tension, as this can easily occur even with an adequate supply of O_2. It can be corrected by gentle pressure on the bag during inspiration, thereby increasing the volume of the respiratory exchange.

Nitrous oxide-pentothal anesthesia has several advantages. For the patient it provides a pleasant induction with a minimum of nausea and vomiting in the postoperative period. It permits the surgeon to use an electric cautery and so materially shortens the time of operation. There is no increased bleeding from vasodilatation, as may occur with other anesthetics. For the anesthetist it makes possible a smooth, rapid induction and allows him rapidly to deepen the anesthesia whenever necessary. It permits frequent aspiration of the bronchial tree without fear of lightening the anesthesia or danger from the release of explosive mixture. It avoids excitement during the periods of induction and recovery. Breathing is quiet, somewhat shallow, and only slightly increased in rate; consequently, it is less likely to dislodge purulent material from the diseased portion of the lung. There is little change in blood pressure or heart rate. Recovery is rapid and patients are cooperative as soon as they begin to react.

Pentothal, however, has certain disadvantages. It is a marked respiratory depressant if used too freely. The period of recovery may be prolonged. Throat reflexes remain active and cough, hiccough, and laryngeal spasm are likely to occur, especially if there is any excess secretion in the trachea or nasopharynx.

Coughing must be avoided as it is probably the most important cause of spread in pulmonary disease. Once it occurs it is not readily controlled by deepening the anesthesia. The offending secretions must be aspirated. Cough, however, can usually be avoided by omitting all preoperative medication and encouraging the patient to continue raising the secretions until he arrives at the operating room. We have found this very important. Premedication in the usual doses given one hour before operation will depress the cough reflex and allow secretions to collect which will give trouble during induction and maintenance of the anesthetic.

After the patient is in position on the operating table morphine sulfate, 1/12 to 1/6 gr., with atropine sulfate, 1/300 to 1/150 gr., is given intravenously using an 18 or 20 gauge needle. The hypodermic syringe is then disconnected and the tubing from the pentothal syringe connected. The needle and tubing are securely fastened in place with adhesive. We prefer an 18 or 20 gauge needle as it is seldom occluded by blood and it can be used to give additional fluids should the need arise. In most cases we have used a 5 per cent solution of pentothal. We prefer atropine to scopolamine as we found that patients receiving scopolamine were likely to be delirious and uncooperative during emergence from anesthesia.

To prevent undue depression of respiration one must be able to inject small amounts of pentothal and avoid giving large doses inadvertently. Various gadgets have been devised for this purpose, but we have used one designed by our hospital engineer, Mr. C. A. Smith. It consists of a spring holder into which a 20 c.c. Luer-Lok syringe slips easily. Behind the plunger is a gear mechanism operated by turning a small handle at the end of the board upon which it is mounted. One complete turn delivers 4/5 c.c. (5 per cent solution) or 40 mg. This arrangement provides pressure on the plunger at all times and prevents blood from entering the needle and clotting. The board is readily clamped to the table or arm board by a "C" clamp.

In thoracoplasties and other similar procedures in which the pleura is not opened, the patient is placed in position for operation. Usually this is on the side with the lower arm extended on an arm board, the head supported by a small pillow slightly below horizontal; the face is turned toward the table and the table in 10 degrees Trendelenburg. This position allows the tongue to fall forward and provides a free airway.

Two or three minutes after the injection of morphine and atropine, induction is started by giving 4/5 c.c. of 5 per cent pentothal solution (one complete turn of the handle). This is repeated at intervals of 5 to 10 seconds till consciousness is lost. The mask of a McKesson Nargraf machine is then strapped in place and 50 per cent nitrous oxide-oxygen mixture administered by semi-closed technique for four or five minutes; thereafter the closed circle with carbon dioxide absorption is employed. Additional pentothal is added as required to maintain a suitable depth of anesthesia. Artificial airways are not used as they tend to stimulate throat reflexes and induce cough or laryngeal spasm. If the patient is supine the chin must be held up. Occasionally the percentage of nitrous oxide has been increased to 70 per cent during closure.

When the pleura is to be opened the patients are intubated using a woven or metal spiral catheter with an inflatable cuff. A curved catheter connector with suction tube nipple is attached to the end of the catheter to permit ready aspiration of the bronchial tree. At first the method of intubation described by Elliot and Arrowood[10] was followed. This required cocainization of the throat and a prophylactic dose of nembutal one hour or more before coming to the operating room. We have found the use of nembutal delays the recovery from anesthesia. In our last 40 cases we have not used cocainization prior to induction. When the lid reflex is lost, 40 to 80 units of Intocostrin are injected intravenously. The laryngoscope is introduced and the cords visualized. Ten per cent cocaine is sprayed directly on the cords and in 10 or 15 seconds the catheter can be readily introduced. One cubic centimeter of 5 per cent cocaine solution is allowed to run down the lumen of the catheter. This quickly controls any coughing which may occur. Catheters are lubricated with Nupercaine or diothane ointment and are well tolerated even with very light anesthesia. In patients with excess secretions as in bronchiectasis or tuberculosis, bronchoscopy is done after induction and thorough aspiration before intubation.

When the pleura is opened, the lung on that side is allowed to collapse and respirations are aided by slight pressure on the bag during inspiration. The

lung is always re-expanded before closure of the chest. If excessive to-and-fro movements of the mediastinum should occur they can be quickly controlled by giving 100 to 150 mg. of pentothal rapidly and deliberately depressing respiration. Adequate exchange is easily maintained by gentle pressure on the bag. There is very little disturbance of blood pressure if blood loss is compensated by blood transfusion. Pulse rate tends to increase 10 to 20 per minute and respirations are usually between 20 and 24, but somewhat shallower than normal.

Five patients received pentothal solution into tissues through the needle tearing a vein or becoming dislodged. These were treated by immediately injecting 2 c.c. of a 1 per cent solution into the area and very little discomfort was afterward experienced by the patient. One patient developed a mild phlebitis at the site of injection. In this case we had chosen a vein which had been used several times for intravenous fluids. We experienced no difficulty with laryngeal spasm. A rather slow induction, adequate oxygen, and attention to expectoration of excess secretions preoperatively lessens the incidence of this complication. Upon return to their rooms most patients receive 1000 c.c. 5 per cent glucose intravenously and 5 to 6 liters of oxygen per minute through a nasal catheter. Six, whose breathing was shallow, were given 3 to 4 c.c. metrazol intravenously with satisfactory results. Usually a patient with secretions will expectorate on command 10 to 15 minutes before fully regaining consciousness.

Patients anesthetized less than one hour receive from 0.3 to 1 Gm. of pentothal and are usually reacting by the time they are returned to their rooms. Patients with pneumonectomies and lobectomies seldom receive more than 1.5 Gm. These patients usually react within one-half hour.

The older patients as a rule take longer to regain consciousness and their blood pressure and pulse changes are somewhat greater.

A tabulation of all deaths, whether from surgery or extension of existing disease, is given in Table I. In no instance do we feel that the anesthetic was a major contributing factor.

Complications were as follows: Thoracoplasties where fair-sized cavities were being collapsed had increased cough for a few days postoperatively. One patient had tracheitis for two weeks following intubation. No cases of atelectasis were diagnosed. All patients who had prolonged postoperative fever were x-rayed. There were four cases of spread, one following pneumonectomy on a very poor-risk patient who died eight weeks postoperatively; one following lobectomy for tuberculosis with death eleven months postoperatively. Two patients with thoracoplasty had evidence of spread which cleared in a few weeks. There was one case of extension of tuberculosis to the right upper lobe following a right lower lobectomy for cavity. This cleared within six weeks.

Gastrointestinal complications were chiefly nausea and vomiting. Thirty per cent of the patients in whom intubation was not done had slight nausea and vomited once the evening of operation, usually after taking fluids. Ten per cent had moderately severe nausea and vomiting for twenty-four to forty-eight hours.

TABLE I.

OPERATION	DISEASE	TIME OF DEATH	CAUSE
Thoracoplasty 2nd stage	T.B.	10th P. O. day	Cardiac failure, syphilitic heart disease
Thoracoplasty 2nd stage	T.B.	3rd P. O. day	Diabetic coma, severe diabetic
Thoracoplasty 2nd stage	T.B.	13th P. O. day	Pulmonary hemorrhage
Revision thoracoplasty	T.B.	4th P. O. day	Cardiac failure
Revision thoracoplasty	T.B.	5th P. O. day	Cardiac failure
Sequestrectomy	Osteomyelitis of rib	14th P. O. day	Meningitis
Lobectomy	T.B.	5 weeks	Developed bronchial fistula, 4 weeks P. O.
Lobectomy	Carcinoma	2 days	Pulmonary edema
Lobectomy	T.B.	11 months	Spread and reactivation of opposite side
Pneumonectomy	T.B.	On table	Cardiac arrest following clamping of main stem bronchus
Pneumonectomy	T.B.	8 weeks	Spread
Pneumonectomy	T.B.	4th P. O. day	Cardiac failure
Pneumonectomy	T.B.	9 weeks	Myocardial failure, amyloidosis
Pneumonectomy	Carcinoma	2nd P.O. day	Embolus to brain, hemiplegia
Pneumonectomy	Carcinoma	On table	Hemorrhage from pulmonary artery
Exploratory and biopsy of mediastinal glands	T.B.	36 hr. P. O.	Myocardial failure
Exploratory	Carcinoma of stomach and esophagus	1 day	Massive hematemesis
Exploratory	Carcinoma of lung with bronchial obstruction	3 days	Myocardial failure following attempt to go to lavatory
Excision of secondary carcinoma involving chest wall and segmental resection of lung	Metastatic carcinoma	21 days	Carcinoma, hypernephroma

This was frequently associated with coughing, narcotics, or ingestion of food. Only three patients had severe persistent vomiting. One of these was a severe diabetic, difficult to control.

Patients in whom intubation was done had less difficulty. Slight nausea and vomiting occurred in 12 per cent of cases. Two had moderate abdominal distention.

Two patients required catheterization postoperatively.

One patient was irrational for two days following one stage of thoracoplasty. Four patients with inoperable carcinoma of the lung who were slightly irrational at times before operation became quite disoriented after operation. These cases can probably be explained on the basis of hypoxia; they came to operation cyanotic, with a bronchus more or less obstructed by the tumor. This prevented a normal exchange of gases in the blood reaching the involved lung. Postoperative elevation of temperature with its increased metabolism might well account for the increase in this symptom. Definite improvement in cyanosis occurs when it is possible to remove the involved lobes. One patient with pneumonectomy became irrational whenever the bladder was distended.

TABLE II

NO.	OPERATION	DISEASE	AGE	GM. OF PENTOTHAL	TIME OF OPERATION (MIN.)	TIME OF RECOVERY (MIN.)	AVERAGE B.P. CHANGE	AVERAGE PULSE CHANGE	RESPIRATORY CHANGE
181	Thoracoplasty 83 patients	Tuberculosis	17-60	.350 to 1.6 (Av. .7)	30-120	5-60 (Av. 20)	−5	+15	+4
24	Thoracotomy	Empyema	22-55	.4 to 1.7 (Av. .7)	15-90	5-60 (Av. 15)	−5	+10	+4
15	Cavernostomy	Tuberculosis	27-38	.5 to 1.5 (Av. .7)	25-75	10-60 (Av. 25)	−5	+10	+4
6	Revision thoracoplasty	Tuberculosis	25-36	.4 to 1.2 (Av. .8)	60-145	10-60 (Av. 25)	−15	+20	+2
7	Extrapleural pneumonolysis	Tuberculosis	18-45	.7 to 1 (Av. .8)	50-75	10-45 (Av. 20)	−5	+15	+4
3	Excision of sequestra	Osteomyelitis of ribs	39-60	.7 to 1.1	20-45	5-30	−5	+10	+5
5	Excision of tumors of chest wall	2 benign, 2 secondary carcinoma, 1 osteochondroma	37-54	.35 to .65	20-75	10-30	0	0	0
4	Partial scapulectomy; post-thoracoplasty	Tuberculosis	25-30	.8 to 1	30-45	10	−10	+20	+4
4	Excision of diverticulum of esophagus,	Diverticulum	55-61	.35 to .75	40-70	10	−10	+20	+4
2	Excision of cervical glands	Carcinoma	51-63	.5 to .8	60-100	30	−15	0	0
35	Lobectomies	26 bronchiectasis, 4 tuberculosis, 1 carcinoma, 3 empyema with abscess, 1 hemoptysis	15-52 21-55 55 35	.7 to 1.7	65-150	15-80 (Av. 35)	−5	+6	+4
2	Laryngofissures	Carcinoma	39, 50	1 to 1.2	40-50	30	0	+25	+4
21	Pneumonectomies	1 benign tumor, 13 carcinoma, 7 tuberculosis	40 41-60 16-40	1 to 2.3	150-300	15-75 (Av. 40)	−10	+10	+2
19	Exploratory thoracotomy	17 carcinoma, 2 tuberculosis	39-61 57	.7 to 1.65	110-200	15-75 (Av. 30)	−15	+10	+2
2	Repair of diaphragmatic hernia	Diaphragmatic hernia	42, 45	1 to 1.4	135	40-90	0	+6	0
43	Miscellaneous								

SUMMARY

We are reporting the use of combined pentothal sodium and nitrous oxide in 363 consecutive cases. From this experience we have arrived at the following conclusions:

1. It provides a high oxygen tension.
2. It is nonexplosive.
3. It is nonirritant to the lungs.
4. It has very little effect on blood pressure or pulse rate.
5. Excitement during induction and recovery is avoided. Respiratory depression, cough, and laryngeal spasm can be minimized and the period of recovery need not be excessively long. Postoperative complications have been few, considering the seriousness of the operations and the physical status of the patients.

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AN ANGIOGRAPHIC STUDY OF THE FORM AND FUNCTION OF THE REMAINING LUNG AFTER PNEUMONECTOMY

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BOTH the form and function of the remaining lung after pneumonectomy have become matters of clinical as well as theoretical interest because of the rapid increase in the number of total pneumonectomies being performed. A survey of the experimental and clinical studies which have been made may begin with reference to the comprehensive work on dogs of Longacre, Carter, and associates.[1, 2] They demonstrated that the age of the animals at the time of pneumonectomy influenced the subsequent behavior of these animals and also the pathologic findings in the remaining lung. In puppies, the functional capacity of these animals under conditions of physical stress soon returned to normal. There was enlargement of the remaining lung, and, microscopically, increase in number and decrease in size of the alveoli. When adult dogs were used, the functional capacity was found to be greatly reduced after operation. The remaining lung was enlarged but there was evidence of emphysema microscopically. When growing dogs were used, there occurred an early return to normal function to be followed some years later by moderate functional reduction. Microscopic study revealed both regenerative and emphysematous processes. These observations are consistent with those of Bremer,[3] who noted that simple distention by dilation of the alveoli and respiratory units occurred after lobectomy or pneumonectomy in cats. However, a true regeneration by means of new growth of normal alveoli and respiratory units may occur. "Regeneration occurs in the young, dilatation in those whose lungs have ceased growing."[3]

Changes in the remaining lung after pneumonectomy in childhood have been described by Lester, Cournand, and Riley,[4] who measured lung volumes, maximum breathing capacity, ventilation, breathing reserve, and respiratory gas exchange under basal conditions, during moderate and severe exercise, and shortly following exercise. Fifteen normal children and three children who had undergone pneumonectomy were used for this study. The results demonstrated that the total capacities of the remaining lungs were smaller in the three who had undergone pneumonectomy than the total capacities of both lungs of normal children, but larger than that of the right lung alone. The vital capacities in each were greater than the assumed vital capacity of the right lung of controls. The functional residual air was found to be smaller in amount in cases where the lung was not pulled into the opposite chest. The dead space in the bronchial tree was reduced and a relatively larger proportion of the tidal air became effective, provided the total volume of tidal air was not reduced.

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Since more blood circulated through the remaining lung, the authors expressed the view that the functional diffusing surface was either increased in area by increase in the number of capillaries, or increased in efficiency by more blood passing through previously existing capillaries. The efficiency of gas exchange between capillaries and alveoli was, therefore, believed to be increased. The breathing reserves were found to be lower in various degrees of activity. However, even in the most exhausting exercise, the reserves were not completely expended. It was suggested that overdistention of the lung following pneumonectomy is not desrable and that planned procedures to eliminate the possibility of marked mediastinal displacement should be considered.

Studies in adults in the immediate postoperative period have been carried out by Maier and Cournand.[5] Determinations of arterial oxygen saturation demonstrated the unreliability of clinical criteria of anoxia. They found that the majority of patients had slight reduction in arterial oxygen saturation during the first postoperative week after pneumonectomy. In two cases of prolonged arterial oxygen unsaturation, there was a mediastinal shift. The return of the mediastinum to the midline accomplished by aspiration of fluid produced a rapid return of the normal oxygen saturation level.

Cournand and Berry[6] investigated adults at a later period after pneumonectomy and found that the chief difference from normal function was "A reduction in breathing reserve in various states of activity. This reduction, caused by a decrease in maximum breathing capacity, was greater in the older patients because of the cumulative effect of abnormal hyperventilation."[6] They also found that "The decrease in maximum breathing capacity was not proportional to the loss of lung volume and was greatly influenced by the state of distention of the remaining lung. The late effects of pneumonectomy upon gas exchange in the lungs, the state of the respiratory gases in the arterial blood, and the cardiocirculatory function, were insignificant."[6] Furthermore, in four patients on whom a supplemental thoracoplasty was performed, the ventilatory function was not adversely affected. In fact, this procedure definitely helped several of these patients. The authors suggested that emphysema does not, perhaps, in itself prevent satisfactory functional results if the remaining lung is not permitted to expand further after operation. They found a greater incidence of dyspnea in the age group 53 to 65, because of the larger reduction in the ability of the chest bellows to circulate air and the larger ventilating requirements during and following moderate exercise. There was also found a reduction in the efficiency of this group to supply oxygen to the tissues during and immediately following exercise.

In Rienhoff's study[7] of twelve patients who had undergone pneumonectomy, "There has been some difference in the degree of compensatory dilatation in the eight patients with total pneumonectomy who survived operation, but in all of them some hyperdistention occurred. This difference in degree seemed not to depend on the age of the patient." He found a marked tracheal displacement in all his cases and concluded that "We have been able to elicit no evidence whatsoever that compensatory dilatation of the lung in either man or animal is harmful."

A few observations may be made on the foregoing outline of the work which has been done on the subject. First, there is as yet no evidence in children of changes in the lung as noted in experiments on young animals. Second, some measure of dilatation of blood vessels and hyperdistention of the lung (emphysema?) is believed to occur with mediastinal displacement, which, according to one set of observations, results in interference with function and, according to another, does not. Third, almost all agree that a pronounced deviation of the mediastinum is the factor which produces compensatory changes in blood vessels and parenchyma and resultant reduction in function, particularly in the older age group. For this reason thoracoplasty is advocated by some, either immediately, to obviate mediastinal displacement, or subsequently, in order to improve existing dyspnea.

Since the important question of alteration in form and function is concerned with the ultimate and not the immediate, and since the severest test would be in the older age group, we chose a substantial number of patients with pneumonectomy performed for cancer for clinical study. The time which had elapsed since operation ranged from six months to ten years. According to clinical tests (vital capacity and dyspnea on mild exertion) significantly reduced pulmonary function was not seen, regardless of deviation of the mediastinum, which was not infrequent and was, at times, pronounced. There were a few exceptions in which the remaining lung was known to have been diseased before operation, or in which it became infected, with the sequelae of infection occurring.

It was then evident that a study of the topography of the pulmonary vessels in these cases would throw additional light on the question of the change in form and perhaps offer indirect evidence as to changes in function. Twelve patients ranging in age from 18 to 62 years, who had undergone pneumonectomy for malignant tumors, were utilized for angiographic studies. They were chosen at random, some with and some without pronounced mediastinal distortion, and they represented varying intervals after pneumonectomy, ranging from six months to ten years. (Chart 1.)

The technique of angiography was that developed by Sussman[8] at the Mount Sinai Hospital. The patient received an intravenous injection of 50 c.c. of 70 per cent Diodrast, and five films were taken in rapid succession by a specially devised casette shifter, so that the various mediastinal and pulmonary vessels were successively visualized. There were no serious untoward reactions even among patients over 60 years of age.

The films were studied with the following questions in mind:

1. Are there any gross alterations in size, shape, or distribution of the vascular pattern in the remaining lung?
2. Are there any significant changes in the mediastinum or its vessels?
3. To what extent does the remaining lung expand, as evidenced by the distribution of blood vessels?
4. Is the time element after pneumonectomy itself responsible for significant vascular changes?
5. What happens to the stump of the severed pulmonary artery?

CHART I.

NAME	AGE	DIAGNOSIS	SIDE OF PNEUMONECTOMY	INTERVAL AFTER PNEUMONECTOMY	MEDIASTINAL SHIFT
J. D.	60	Squamous cell carcinoma	Right	6 mo.	Moderate
J. E.	60	Squamous cell carcinoma	Left	8 mo.	None
J. Q.	60	Squamous cell carcinoma	Left	8 mo.	Moderate
I. W.	62	Squamous cell carcinoma	Right	10 mo.	None (thoracoplasty)
A. B.	47	Cylindroma	Right	1½ yr.	None (thoracoplasty)
A. F.	55	Squamous cell carcinoma	Right	2 yr.	Slight
A. B.	63	Squamous cell carcinoma	Right	2 yr.	Marked
J. W.	58	Squamous cell carcinoma	Right	2½ yr.	None (thoracoplasty)
L. R.	48	Mucous cell carcinoma	Right	3 yr.	Moderate
E. S.	18	Adenoma with invasion of lymph nodes	Right	4 yr.	Slight
R. K.	58	Squamous cell carcinoma	Left	7 yr.	Moderate
A. C.	50	Sarcoma	Left	10 yr.	Moderate

As far as any gross alteration of vascular pattern in the remaining lung is concerned, it was seen that there was a notable uniformity of findings among almost all the patients. The vascular pattern presented an essentially normal arrangement, in regard to size of vessels and their distribution. There were some minor abnormalities, and these consisted of:

1. Varying degrees of displacement of the pulmonary artery toward the operated side, accompanying the mediastinum
2. Some angulation and a turning on itself of the uppermost medial branch of the remaining pulmonary artery
3. Visibility of the vessels at a lower level than usual in the lower lobe, delineating a downward displacement of the diaphragm.

These features are well illustrated in Fig. 1: This was a 47-year-old woman who had had a right pneumonectomy three years previously for diffuse mucous-cell carcinoma. Her postoperative course was uneventful and she has remained asymptomatic since operation, complaining of shortness of breath only when performing strenuous activity. Her angiogram revealed a moderate shift of the mediastinum toward the operated side, and demonstrated clearly how the uppermost branch of the remaining pulmonary artery may become "turned on itself" to supply that portion of the lung which has "bulged" into the right side of the chest. This vascular distortion, although present in all cases to some degree, was marked in this patient. It can also be seen in Fig. 1 that the vessels in the lower lobe and the pulmonary parenchyma itself are prominent at a lower level than usual, associated with descent of the diaphragm. The vessels can be seen behind the gastric "air bubble." Aside from these changes, the vascular pattern is within the normal range.

In only one patient did the remaining pulmonary artery appear to be somewhat larger than normal. This was in a 58-year-old patient who was operated upon seven years previously. Even in this patient one cannot be sure of enlargement of the vessel. In the one case in which angiograms were performed before pneumonectomy, there was no significant change in the size of the remaining pulmonary artery when the first angiograms were compared with ones taken six months after operation.

Fig. 1.—Three years after pneumonectomy. Mediastinum moderately shifted toward operated side. Curved superior branch of left pulmonary artery with branches crossing midline, indicates expansion of upper lobe into dead space. Downward expansion of lower lobe visible.

Concerning the major mediastinal vessels, it can be anticipated from Fig. 1, that with this moderate mediastinal displacement, there is no significant displacement of the aorta. This was found to be true in the vast majority of cases. However, the large venous trunks are distorted in some cases, as seen in Fig. 3, in which there is to be seen pronounced displacement of the left innominate vein and superior vena cava. This patient was a 63-year-old man,

seen two years after pneumonectomy for carcinoma. The postoperative course was essentially uneventful, but he developed the most severe mediastinal shift of all the cases in this series. Even with this marked change in position, there is no significant obstructive element or irregularity in the lumina of the affected vessels, and the pattern of the pulmonary artery and branches in the remaining lung is essentially normal. Hence, no disturbance in cardiopulmonary function need be anticipated from the films, and none, in fact, exists clinically.

Fig. 2.—Seven years after pneumonectomy. Pulmonary vascular pattern essentially normal except for probable enlargement of main pulmonary artery.

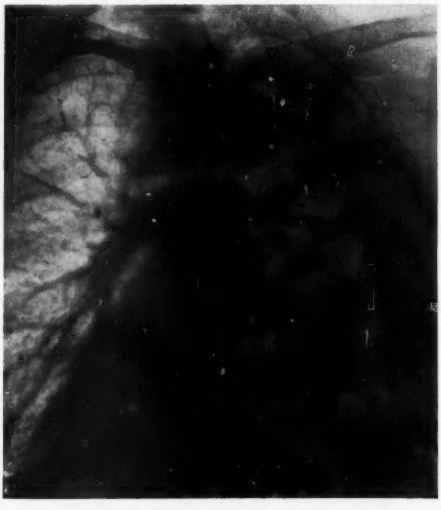

Fig. 3.—Two years after pneumonectomy. Left innominate vein and superior vena cava extremely displaced without symptomatic effect. Normal vascular pattern in remaining lung.

Turning to a consideration of the remaining lung, in this group of cases it is evident from the angiograms that extreme hyperdistention has not occurred even with pronounced distortion of the mediastinum. A portion of the upper lobe "bulges" into the operative area along with the tracheal shift, when mediastinal displacement occurs, as seen in Fig. 1. Judging from the pulmonary vessels within the bulge, the amount of pulmonary tissue involved is not great. In any event the vessels within the bulge are not greatly distorted or angulated and are neither thinned nor widely separated as would be anticipated if pulmonary emphysema occurred in this area.

Fig. 4.—Eight months after pneumonectomy. Posterior-anterior erect film. Elevation of diaphragm demonstrates largely obliterated dead space.

A consistent feature is the downward displacement of the diaphragm which appears to be due to an expansion of the lower lobe rather than the lung as a whole, to judge from angiograms. Often enough it is only in this direction that the lung can expand, for the dead space on the operated side may become rapidly reduced by the presence of fluid and the upward shift of the diaphragm (Fig. 4).

The important question is whether or not the expansion of the lung in any direction occurs at the expense of function (emphysema) with "compensatory" changes in the vascular pattern and (or) parenchyma. The angiograms appear to establish that simple expansion occurs. In this connection it may not be amiss to emphasize the proved existence of a considerable reserve in the

normal lung and that large areas in the lower lobes function only to a minimal extent except on violent exertion. Thus, the enlargement of the lung after pneumonectomy may represent nothing more than an unfolding or opening up, so to speak, of these areas. The angiograms appear to establish this point for the branches of the pulmonary artery are spread over a larger area, particularly in the lower lobes, but they are unaltered in diameter. Any significant degree of emphysema would probably produce changes in the vascular pattern, according to the classic work of Robb and Steinberg.[9] They demonstrated by angiography that in emphysema, the main branches of the pulmonary artery appear enlarged and the peripheral branches narrowed and wirelike.

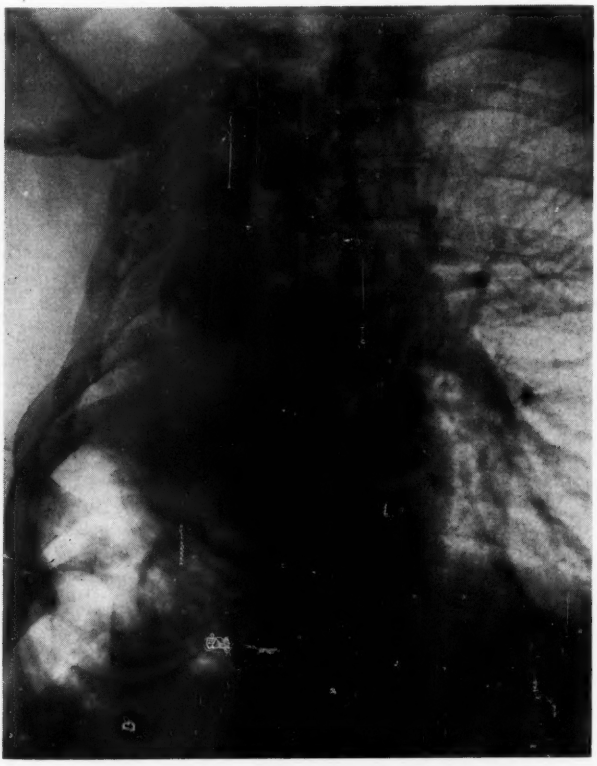

Fig. 5.—Ten years after pneumonectomy. Patient asymptomatic despite displacement of heart and great vessels. Normal vascular pattern in remaining lung.

Concerning changes in the vascular pattern in relationship to the age of the patient and the interval of time since pneumonectomy, the youngest patient in this series was 14 years old at the time of pneumonectomy. The angiograms four years postoperatively revealed the vessels in the remaining lung to be as completely normal as in the older age groups. It was felt that perhaps if a long enough period elapsed after pneumonectomy, there might develop gross

vascular changes. In Fig. 5, which is an angiogram performed ten years after pneumonectomy for primary sarcoma of the lung, when the patient was 40 years old, the pulmonary vascular pattern appears to be normal. In this connection it should be mentioned again that in the one patient in this series who did seem to have a somewhat enlarged pulmonary artery, the time interval after pneumonectomy was seven years.

The condition of the stump of the severed pulmonary artery can be considered briefly. In many cases it was clearly visualized and in no instance was there any enlargement or suggestion of thrombus formation. In Fig. 6, which is an angiogram two years postpneumonectomy in a 55-year-old patient, the stump is well visualized. The clips on the adjacent main bronchus are seen, a technique for closure of the bronchus which was employed occasionally at that period.

Fig. 6.—Two years after pneumonectomy. Smooth stump of pulmonary artery to be noted below metal clips on bronchial stump. Vascular pattern normal.

A brief discussion concerning the interpretation of angiograms is in order before stating the conclusions which have been reached from this study. In the first place, this appears to be the first investigation by angiography of the remaining lung, and the number of cases is not large. Second, the interpretation of topography by angiograms is direct but the interpretation of function is inferential. Finally, vascular alterations in the finer branches of the pulmonary artery may be present but cannot be safely interpreted by the angiographic technique which has been employed. The position of the patient, the phase of

respiration, the position of the diaphragm, and the physique of the patient are factors which must be considered in the analysis of the films and which render unsafe the interpretation of changes, if any, in the finer branches of the pulmonary artery.

CONCLUSIONS

1. There is no significant abnormality in size, shape, or distribution, of the vascular pattern in the remaining lung after pneumonectomy, in the majority of cases.

2. In this series, when no thoracoplasty had been performed, there often occurred moderate displacement of the mediastinum and great vessels; this was marked in only one case.

3. The expansion of the remaining lung, as judged by the vascular pattern, was not as great in these cases as has been described.

4. There occurs no alteration in size or recognizeable thrombosis within the stump of the resected pulmonary artery.

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GASTRIC CYST OF THE MEDIASTINUM

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ALTHOUGH gastric cysts of the mediastinum are of infrequent occurrence, the serious effects produced by them warrant consideration of the knowledge gained by study of even single cases.

Excellent reviews of reported cases have been made by Olken,[21] by Olenik and Tandatnick,[20] and by Schwartz and Williams.[25] There are, in addition, several cases that have not been included in the reviews of these authors. An appropriate perspective may be gained by a summary of the cases reported to date. Twenty-seven cases[1-32] have been reported and usually no one person's experience has embraced more than a single case. Of the twenty-seven reported cases only two were found in individuals over 15 years of age; nineteen were in infants under 1 year of age; four were between 12 and 24 months of age; and one child was 4 years old. Fourteen were males, ten females, and in three the sex was not stated. From these figures it is apparent that the anomaly is indeed an unusual one and that it is usually of clinical significance in the first year of life.

Terminologic idiosyncrasies and inaccuracies have caused considerable confusion. Cysts containing gastric mucosa have been called gastric cysts, gastrogenic cysts, accessory stomach, mediastinal cysts of gastric origin, thoracic gastric cysts, esophageal duplications, mediastinal cysts of enteric origin, and gastroenteric cysts. In our opinion simplicity and accuracy are served equally well by calling these structures gastric cysts of the mediastinum since it is the gastric mucosa which produces a course different from other mediastinal cysts. That they are cysts containing fluid is without doubt. It is equally true that all contain some elements of the gastric wall; mucosa only in some, all three layers in others. In some the gastric origin of the cyst is recognizable only by a small fragment of mucosa resembling that of the normal stomach. In others the cyst in its entirety may be a facsimile of the normal stomach. This is true in our own case and in the cases reported by several others.[16, 17, 25, 32]

So little mucosa may be present that its secretory nature is not apparent or the mucosa may secrete copious quantities of gastric juice containing hydrochloric acid, pepsin, and rennin.

In infants and children gastric cysts, if at all productive of symptoms, are usually rather rapidly lethal unless they are removed. In adults the difficulties of differential diagnosis among benign cysts of the mediastinum and malignant or potentially malignant tumors are so great that excision is usually desirable and advisable.

THEORIES OF DEVELOPMENT

The entire problem of congenital defects and abnormalities remains considerably shrouded in obscurity despite several explanations that have been offered. It is possible to understand what has happened to produce these ab-

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normalities; however, it is not understood why this deviation from normal has taken place or precisely what physiologic or physical aberrations have occurred in the embryo to produce the pathologic state in the infant.

Several hypotheses have been offered to explain the occurrence of cystic tumors of the mediastinum. Lewis and Thyng[15] have noted that knoblike intestinal diverticula are found frequently in embryos of the pig, rabbit, and man. They believe that from these structures there may arise diverticula which usually degenerate and disappear but which occasionally may persist as permanent diverticula or cysts. This observation would, in part, explain the common occurrence of multiple cysts randomly situated along the gastrointestinal tract and the histologic similarity of mediastinal cysts and intestinal reduplications and cysts found by Ladd and Gross[12] and by Poncher and Milles.[22] The latter authors have described the post-mortem findings in an infant 29 months of age in whom there were found an intramesenteric diverticulum arising from the ileum and containing in its wall gastric mucosa and a polyp composed of gastric mucosa and two extrapleural cysts of the mediastinum which contained gastric mucosa, most of which had undergone pressure atrophy and perhaps digestive necrosis.

The findings of Lewis and Thyng perhaps would explain the origin of the mediastinal cyst in the case reported by Steele and Schmitz.[29] In their patient a cystic tumor was removed from the posterior mediastinum and a firm nodule measuring 2 by 1 cm. was found at the lower pole. The nodule was covered by mucosa which resembled that of the fundus of the stomach.

Nevertheless, it is necessary to go beyond this hypothesis to explain the cystic tumors of the mediastinum which are almost complete anatomic and physiologic reduplications of the normal stomach. In such cases there develops within the mediastinum what is virtually an accessory stomach containing mucosa, submucosa, and muscular layers entirely similar to those of the normal stomach and carrying out a functional activity that has in some resulted in peptic ulceration of the wall of the tumor. Such cysts are found only in infants since gastric juices are produced rapidly and retained in a closed space so that the increasing size of the cyst soon causes death by interference with respiratory function, or by rupture into a neighboring organ.

Needham[18] has reviewed the knowledge and theories of functional development of the embryo. From this review one gathers that there is an organizer-hormone (an evocator) present throughout the egg. The organizer is believed to determine the eventual form of cells which it influences. Moreover, the organizer will influence similar cells in a different manner according to their locations.

Schwartz and Williams[25] have utilized the material gathered by Needham in his general review of the subject and have synthesized a theory to explain the development of gastric cysts within the mediastinum. They suggest that gastric cysts develop by this mechanism: "The organiser substance that causes the evagination of the lung buds and their further proliferation, either through some change in time or intensity factors along its gradient causes the evagination of what would form an accessory lung (Nebenlungen) in the region of the cardia of the stomach or lower esophagus. Because of the anatomic situation

at the time this could occur, the assumption seems reasonable. As the result of changes in the anatomic relationships that accompany continued development, this organiser influence wanes thus allowing the stomach organisers to exert their influence to the completion of differential development even to include functional activity. With the formation of the mesenchymal coats (later muscular) and the development of the diaphragm, this exclusion is pinched off into the posterior mediastinum, but still retains sufficient attachment to the lower end of the esophagus to rotate to the right with the rotation of the stomach."

This hypothesis has certain convenient aspects but remains "an assumption" and does not offer much information as to why these things happen or how. It must be admitted that although interruption of the orderly development of the germ layers does occur and by this occurrence does produce abnormalities and defects in the infant, the fundamental reason for such occurrence is not known.

Understanding of the results produced by congenital abnormalities is on much sounder ground. It is well appreciated that developmental aberrations of the embryo may produce pathologic states in the infant and adult that are of varying symptomatic magnitude. Thus a mediastinal cyst may seriously interfere with health and growth and even be a cause of death; or it may remain asymptomatic and be found by chance in an x-ray survey or at post-mortem.

Symptoms produced by these cysts result from pressure on neighboring structures and consequent interference with the functions of these organs or by functional activity within the cyst itself. Symptoms are of earlier occurrence and of greater magnitude in cysts with functional activity. The more nearly the structure of the cyst approximates that of the normal stomach, the more serious will be the symptoms caused by it.

Inasmuch as gastric cysts usually arise in the posterior mediastinum and encroach by their growth upon one or the other pleural space (usually the right), the pressure of their presence first will be apparent on the soft structures of the immediate vicinity. Among twenty-seven reported cases the cyst protruded into the right hemithorax in twenty-one cases; this is probably because the aorta is a fairly effective barrier against extension into the left hemithorax.

Interference with respiratory function is observed most commonly and consequently *dyspnea* is a frequent symptom; this results from pressure on the membranous posterior wall of the trachea and major bronchi and by interference with complete and free expansion of the lungs both by usurpation of the space provided for them and by production of atelectasis as a result of partial or complete bronchial occlusion.

Cough, usually dry and hacking, is caused by irritation of the contiguous trachea and bronchi. The cough may be productive if infection ensues distal to a point of partial bronchial obstruction.

Dysphagia in its true character of difficult swallowing is most uncommon since the liquid and soft foods of the infant diet will pass with ease through even a partially constricted esophagus. However, it is often observed that

dyspnea is markedly increased during feeding and that swallowing may produce transient cyanosis. The result will be a serious interference with nutrition and a refusal to eat when deglution interferes with breathing.

Cyanosis, either transient during feeding, crying, and straining, or persistent, follows increased impairment of respiratory function that may be caused by increase in the size of the cyst or by concomitant atelectasis or pneumonia.

Cardiac palpitation may occur if the cyst involves by pressure the heart or the cardiac nerves.

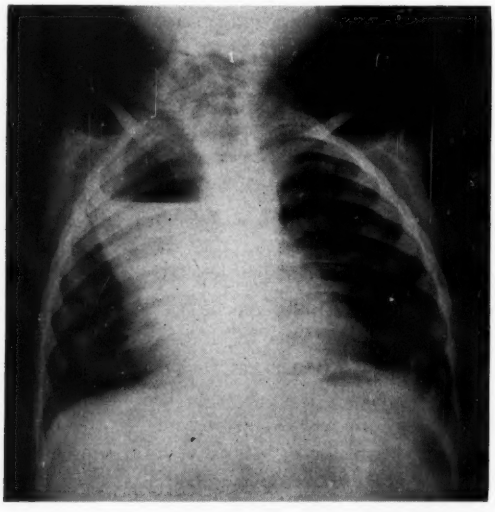

Fig. 1.—Posteroanterior roentgenogram showing fluid level on right side; note hemivertebrae in upper thoracic and cervical spine.

Hemoptysis has been observed occasionally and is caused by actual necrosis of the lung, by involvement of the bronchial vessels, or by the development of a fistula between the cyst and the air passages.

Chest pain occasionally occurs and is probably caused by pressure upon the parietal pleura and intercostal nerves.

Functional activity of the cyst may occur but will cause no symptoms other than hematemesis and then only if there is a communication with the esophagus. *Hematemesis* may be the presenting symptom and be particularly severe if there is peptic ulceration of the cyst wall. It is, of course, possible that in those cases with peptic ulceration of the cyst wall pain may ensue but this is conjectural only.

Melena, either as frank blood in the stools or in the form of tarry stools, may occur if there is a communication between the cyst and the esophagus. Ulcers of the cyst wall or a diffuse inflammatory reaction may cause bleeding and the discharge of blood in vomitus and stools.

Physical findings are those of a space-occupying mass within the chest together with those of the associated pneumonia or atelectasis. However, even a very large cyst may be associated with a surprising paucity of physical signs.

Roentgenograms are indispensable in diagnosis of a mediastinal tumor but there are no roentgen signs that will lead unerringly to a precise diagnosis of a gastric cyst. The appearance on the roentgenogram is that of almost any solid or fluid-containing mediastinal tumor. The dense shadow of the gastric cyst is found in the posterior mediastinum and may extend into the right or left hemithorax. The cyst may contain air if there is a communication with the esophagus or trachea (Figs. 1 and 2). The form of the cyst tends to be spherical in the smaller ones and elongated in the long axis of the body in the larger cysts. The form is, of course, determined by the pliancy or immobility

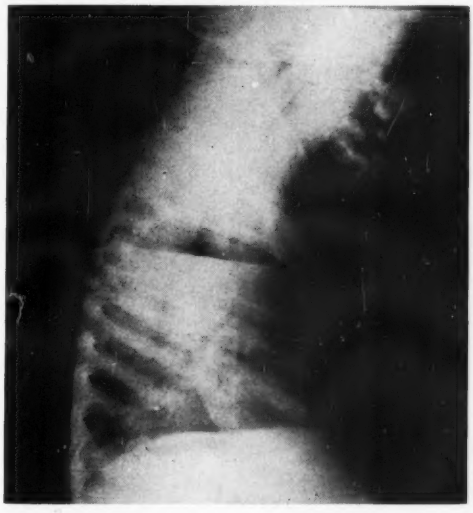

Fig. 2.—Lateral roentgenogram; fluid lies posteriorly.

of the neighboring structures. Ancillary signs of the pressure effects of the cyst are varying degrees of atelectasis and obstructive or compensatory emphysema of the lung. Roentgenograms should be made in the upright position in frontal and lateral projections; oblique projections are occasionally useful in demonstrating smaller cysts. Esophagograms are of great value since displacement or distortion of the relatively mobile and pliant esophagus can be demonstrated readily without risk to the patient. There is little reason for using a bronchogram as a diagnostic aid since this procedure is very difficult in infants and is hazardous as well in those with respiratory embarrassment. Small cysts that cannot be demonstrated in conventional roentgenograms sometimes may be revealed by planigrams of the mediastinum.

Bronchoscopy and esophagoscopy may be helpful in excluding an endobronchial or endoesophageal lesion if such doubt exists; otherwise these examinations will only confirm the knowledge that there is a reduction in the lumen of the tube caused by an extramural mass.

All these diagnostic aids will lead only to the recognition of a probably cystic tumor of the posterior mediastinum without disclosing the true nature of the lesion. Indeed such accuracy of diagnosis, while pleasurable to the physi-

cian and surgeon, is scarcely necessary since almost all cystic tumors of the mediastinum should be removed surgically unless there is a serious contraindication.

The diagnosis may be made absolute by aspiration of fluid from the cyst and demonstration that the fluid is indeed similar to that produced in the stomach. This may be done by titration of its acidity and by the identification of pepsin or rennin. However, aspiration is not without danger of infection and leakage of corrosive gastric juice into the mediastinum or pleural cavity. It should, therefore, be done not to establish a precise pathologic diagnosis of the exact nature of a mediastinal cyst but only if doubt exists that the lesion is a cyst rather than a loculated pleural effusion. In the event that aspiration is deemed essential to diagnosis, the appropriate site for introduction of the needle should be determined by careful localization of the density in roentgenograms taken in at least two projections. The fluid found within the cyst may vary from thin and watery to thick creamy material that resembles pus. However, even a physical resemblance to pus seldom will confirm a diagnosis of empyema since usually the cellular content of the fluid will little resemble that of pus.

TREATMENT

The only effective method of treatment of gastric cysts of the mediastinum is surgical. Complete removal of the cyst in one stage is by far the most desirable method but occasionally expedience will dictate partial removal and destruction of the remaining mucosa or marsupialization with prolonged drainage and repeated efforts to destroy the mucosa. Certainly primary external drainage by thoracotomy or intercostal catheter is illogical and undesirable since the secreting mucosa is not destroyed by this method and eventually excision will have to be attempted through an infected field.

In infants the effects of the cyst are usually so severe as to jeopardize the life of the child and consequently removal of the cyst is imperative. With anesthesia adequate for intrathoracic operations there is no reason why the operation cannot be undertaken even in newborn infants. A transpleural approach through the side on which the cyst is most apparent allows generous exposure of the mediastium and permits the surgeon to dissect the cyst free from its often intimate attachments to the great vessels, the esophagus, and the lung. It would seem that an extrapleural mediastinotomy is unnecessarily restricting in exposure since the danger of an open pleura can readily be overcome by a competent anesthetist.

In adults gastric cysts are of infrequent occurrence and cause severe or life-threatening symptoms since they rarely contain actively secreting gastric mucosa. The rarity of gastric cysts in adults is, of course, an indication of the almost uniformly lethal nature of the anomaly in infancy. Asymptomatic cysts may very occasionally be found in routine x-ray surveys. Blades[2] has reported a series of 109 mediastinal tumors found in adults; although 34 of this number were benign epithelial cysts none contained gastric mucosa. Nevertheless, because of the great difficulty in determining the nature of a mediastinal tumor without histologic examination, removal of the tumor is advisable even though

the patient is in perfect health and without symptoms at the time it is found. In such a case complete removal is most desirable. Occasionally it may be permissible, and indeed sometimes essential, to leave behind a portion of the cyst wall if its removal from the great vessels or other contiguous structures would be too hazardous. In such circumstances the mucosa itself may often be removed or destroyed by an escharotic agent after the cyst has been opened.

Fig. 3.—External appearance of cyst in its distended state; note similarity to normal stomach with pylorus at lower end.

In seventeen of the reported cases operative procedures were carried out; these have been reported by thirteen authors.[3, 5, 6, 12, 13, 16, 17, 19, 22, 25, 29, 31, 32] Complete excision, either as a primary procedure or secondary to drainage or marsupialization of the cyst, was attempted or carried out in twelve cases; of this number seven patients survived and five died as a result of the operation. In five of the cases other operative procedures (exploration alone, marsupialization and cautery destruction of the mucosa, paraffin plombage) were done; of this group two patients survived and three died. It is to be noted that of the seven patients who recovered after excision, two were adults with cysts in which only remnants of gastric mucosa were found.

It is apparent from these figures that operation is hazardous. On the other hand, without operation only a very few infants will survive to adult life with

a gastric cyst and none will survive with cysts containing actively secreting mucosa. From the fact that the successful cases have increased in recent years, it is apparent that, with increasing competence of thoracic surgeons and anesthetists, extensive intrapleural operations in infants can be undertaken more safely.

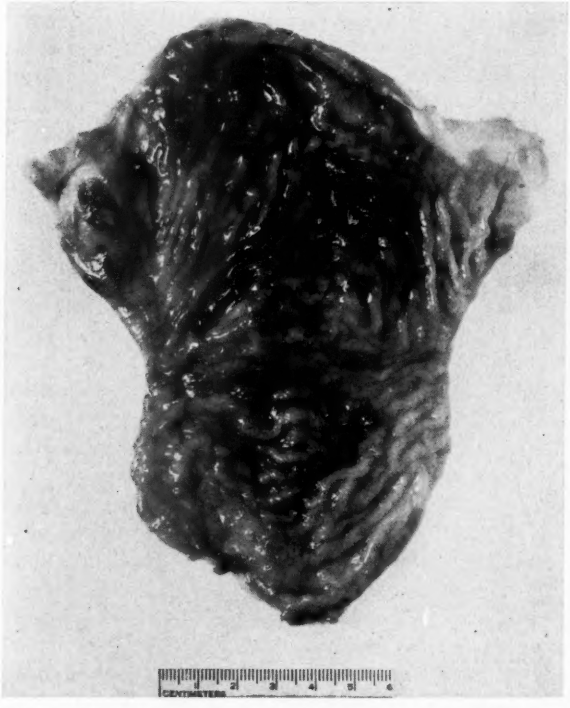

Fig. 4.—Interior of opened cyst; note gastric rugae and large ulcer at upper end.

CASE REPORT

C. M., aged 15 months, male. This child was delivered normally. Development was uneventful for nine months when he had moderate difficulty in eating and a slight cough. During the eleventh month he passed several tarry stools and soon thereafter vomited blood-streaked material. The next day he had a massive hemorrhage with bright red blood pouring from the mouth and rectum. He was hospitalized and given six whole blood transfusions. At that time a roentgenogram of the chest showed an area of increased density in the right hemithorax but the significance of this was not realized. A diagnosis of peptic ulcer was made at another hospital and the child was allowed to return home on a fluid diet. He continued to gain weight but was irritable, coughed frequently, and occasionally had difficulty in breathing in a recumbent position. He seemed to prefer lying on the right side.

At the age of 14 months he was acutely ill with fever, vomiting, diarrhea, and abdominal pain. Roentgenograms taken after this episode had subsided showed the density in the right hemithorax still present but with a fluid level now apparent. Gastrointestinal x-rays and a barium enema were normal. At the time of admission to the Albany Hospital on Feb. 6, 1947, physical examination was normal except for the chest. Expansion

was equal bilaterally, the percussion note was resonant throughout, and breath sounds were bronchovesicular with numerous moist râles and rhonchi. The adventitial sounds were more marked at the right base. He had at that time a mild upper respiratory infection. Urinalysis was normal; the red blood cell count was 4,370,000 with 17 Gm. of hemoglobin; the white blood cell count was 31,500 with 65 per cent polymorphonuclear leucocytes. Roentgenograms of the chest showed an increased density through the upper portion of the right pulmonary field extending downward close to the cardiac shadow to the diaphragm with a

Fig. 5.—Low-power photomicrogram showing mucosa, submucosa, and two muscle layers of cyst wall; note similarity to normal stomach.

fluid level opposite the first anterior rib. In the lateral projection, the greatest opacity was posterior (Figs. 1 and 2). Incidental findings were spina bifida of the lower cervical vertebra and a hemivertebra in the upper thoracic region. Fluoroscopic examination of the chest showed both leaves of the diaphragm to be moving normally. A barium meal failed to reveal any communication between the esophagus and the cystic tumor in the mediastinum. The stomach was normal in position and appearance.

Possible diagnosis of a cystic tumor of the mediastinum or a loculated empyema with a bronchopleural fistula were considered and it was felt that thoracentesis was advisable since ultimate treatment would depend to a large extent on the nature of the fluid. Fifty cubic centimeters of thin, brownish fluid containing darker solid particles were aspirated. This fluid gave a positive guiac test and had a free acid of 12 degrees and a total acid of

23 degrees. Tests for rennin and pepsin were not done. The nature of the fluid at once confirmed a diagnosis of gastric cyst of the mediastinum and surgical removal was decided upon.

Preoperatively the patient was given a transfusion of whole blood and received penicillin for several days. On Feb. 18, 1947, a thoracotomy was done under endotracheal anesthesia. The right sixth rib was removed subperiosteally and the tumor exposed through a transpeural approach. The lung appeared normal although partially collapsed and was free of adhesions except for the apical portion of the upper lobe which was adherent to the mediastinal pleura overlying the cyst. It extended from a point high in the neck to the diaphragm, where it ended in a firm cord. The blood supply was derived from the mediastinum and directly from the aorta. The esophagus was identified and found to lie to the left and anterior to the cyst. No connection could be found between the cyst and the esophagus, although one may have existed high in the neck. The mediastinal pleura was

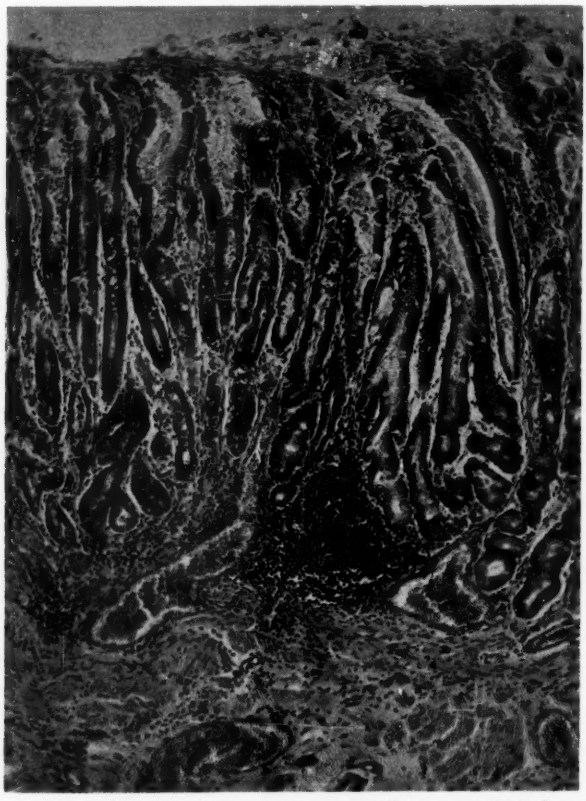

Fig. 6.—High-power magnification of mucosa of cyst wall to show typical secreting gastric mucosa.

incised and the cyst freed by combined sharp and blunt dissection. The mucosa was completely removed from the pedicle which extended into the neck, and the pedicle itself was then ligated with silk sutures. The pleural cavity was lavaged with copious amounts of saline solution and the mediastinal pleura closed loosely with interrupted sutures. The lung was expanded and a small Pezzer catheter was placed through the ninth interspace posteriorly for postoperative drainage. Penicillin, 200,000 units, was instilled into the pleural cavity and the wound closed in layers with interrupted silk sutures.

The cyst measured 14 by 5 by 1.5 cm. in size and resembled grossly a normal infant stomach. The wall was uniformly resilient and averaged .3 cm. in thickness. The inner surface consisted of mucosa in rugal folds typical of stomach. A .5 cm. ulcer was found at the upper end of the cyst. The cyst contained about 75 c.c. of yellowish fluid which was negative for occult blood and had 18.6 degrees free and 32.4 degrees total acid (Figs. 3 and 4).

Microscopic Descriptions.—Sections from various portions of the wall of the specimen showed a typical secreting gastric mucosa. In several situations collections of acute inflammatory cells were scattered throughout the mucosal glands. A small mucosal ulcer covered by fibrinous exudate was present. Mucosal capillaries were congested. The submucosal tissues were slightly fibrosed. The inner musculature, circular layer, was composed of a histologically normal, smooth muscle. The outer musculature, the longitudinal layer, did not appear unusual and was slightly thicker than the inner circular layer. The outer tissue, the serosa, was fibrosed and focally hemorrhagic. Arteries and arterioles in all layers of this cystic structure were not unusual (Figs. 5 and 6).

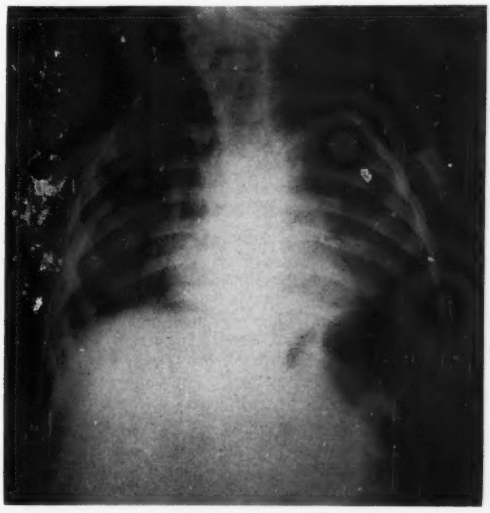

Fig. 7.—Postoperative roentgenogram showing only slight residual pleural thickening.

Diagnosis.—Diagnosis was gastric cyst of mediastinum with a small ulceration of mucosa.

The postoperative course was complicated by development of a pleural effusion on the right side but this responded to frequent aspirations and chemotherapy.

Six months after operation the boy was in good health with no indication of other cysts below the diaphragm. Postoperative roentgenograms (Fig. 7) showed only slight pleural thickening.

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SOLITARY XANTHOMA OF THE LUNG

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WE have recently had an opportunity to observe a clinically benign tumor of the lung in a young woman. After a successful lobectomy with removal of the lesion, pathologic study showed this tumor to be a so-called "xanthoma." Since we have been unable to find a similar case previously recorded, this case is reported in detail.

CASE REPORT

F. M., a 34-year-old white housewife, first came to the outpatient department of the Johns Hopkins Hospital on April 3, 1946, for examination as recommended by her family physician.

Family history was noncontributory. The patient's past health had been good. Ten years previously she had had an episode of albuminuria and edema of the face. This was not associated with pregnancy and cleared without specific therapy in about ten days. Eleven years prior to admission she had an attack of mild left-sided pleurisy lasting about one week and unassociated with cough, fever, weight loss, or hemoptysis. For the past five years she had had occasional ankle edema after standing. The patient denied any exposure to tuberculosis.

The present illness was dated from September, 1945, at which time she had mild hemoptysis for five days. This was intermittent and associated with pain in the right supraclavicular region. It was not associated with fever, sweats, weight loss, or malaise. Shortly after this a chest x-ray taken by the family physician is said to have shown a lesion in the lung, not thought to be tuberculous.

Since September, 1945, the patient had been completely asymptomatic; there had been no further hemoptysis, and no pain, cough, weight loss, or sweats.

Physical examination upon admission to the outpatient department was completely negative except for slight impairment of breath sounds over the right lower chest.

A chest x-ray taken at this time (Fig. 1) revealed a normal cardiovascular shadow. In the right cardiophrenic angle a circular circumscribed lesion was apparent. On fluoroscopy this was found to be in the anterior portion of the right lower lobe. The remainder of the lung fields was clear. Bronchoscopy on two occasions failed to show any endobronchial pathology. Bronchograms showed no communication of the mass with any bronchus and were normal in all other respects (Fig. 2).

A tuberculous lesion was strongly suspected and for this reason the patient was followed carefully for a period of ten months. Serial x-rays during this period did not show any change in the size, shape, or position of the mass. Repeated smears and cultures of the sputum failed to reveal tubercle bacilli. The Mantoux test was only slightly positive after forty-eight hours with 0.1 c.c. of 1:1,000 old tuberculin.

Accordingly a diagnosis of lung tumor, probably benign, was made and on Feb. 3, 1947, the patient was admitted to the surgical service of this hospital.

During the period of observation in the out-patient department the patient had remained entirely asymptomatic.

Physical Examination.—The temperature was 99.2° F.; pulse, 88; respiration, 22; blood pressure, 96/70.

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The patient was a well-developed, well-nourished, white woman who did not appear to be ill. There was no cyanosis. Examination of the head, eyes, ears, nose, and throat was essentially negative. The heart was not enlarged to percussion and no murmurs were heard. Radial pulses were equal and regular. The chest moved equally and adequately with respiration. The percussion note was everywhere resonant and the lung bases descended fully and equally. The breath sounds were vesicular throughout but slightly distant over the right

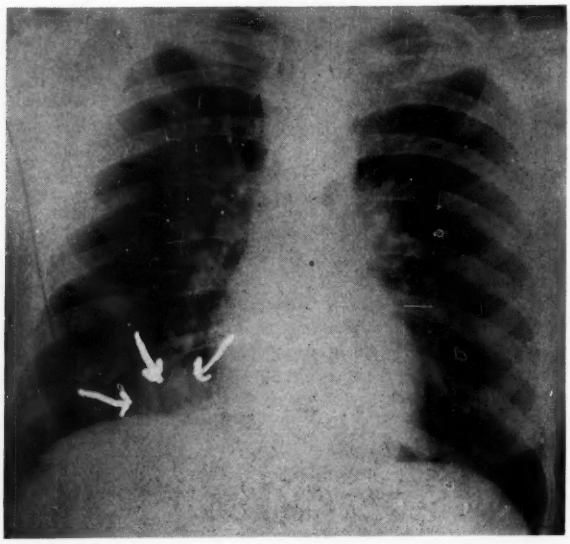

Fig. 1.—Roentgenogram of chest in patient with solitary xanthoma of lung.

Fig. 2.—Bronchogram demonstrating xanthoma in right lower lobe of lung.

lower chest posteriorly. There was no clubbing of fingers or toes. The remainder of the physical examination revealed no abnormalities.

Laboratory Studies.—Urinalysis was negative; hemoglobin 13 grams per cent; leucocyte count was 6,000 with normal differential; sedimentation rate (corrected) 24.0 mm. per hour; icteric index 6.0; sputum smear revealed no acid-fast organisms.

Operation.—On Feb. 5, 1947, Dr. Alfred Blalock, using endotracheal cyclopropane-ether anesthesia, performed a thoracotomy through a right posterolateral incision, resecting a portion of the sixth rib posteriorly. A few filmy adhesions were present between the lung and the chest wall. The upper and middle lobes were normal in appearance and consistency. The basal segment of the right lower lobe contained a hard mass the size of a large lemon which was completely contained within the parenchyma of the lobe. No other masses were palpable and no hilar nodes were felt. Accordingly a right lower lobectomy was performed and the chest closed without drainage. The patient withstood the procedure well.

Course.—The postoperative course was extremely smooth. The patient was ambulatory and comfortable by the second postoperative day. X-rays revealed good expansion of the remaining lobes with a minimum of fluid. Thoracentesis was not necessary. She was given penicillin 240,000 units daily for nine days.

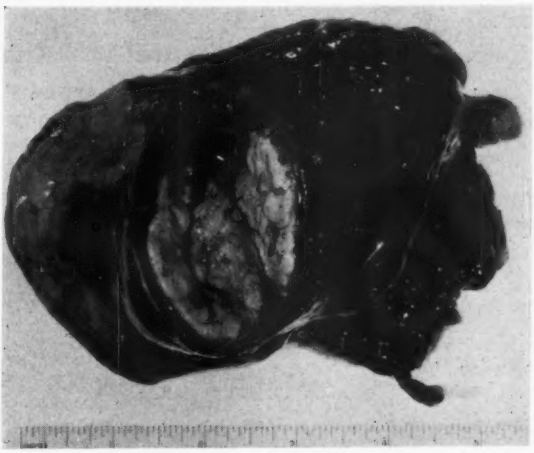

Fig. 3.—Photograph of resected right lower lobe (patient F. M.) with incision into substance of lobe exposing well-circumscribed xanthoma.

Because of the xanthomatous appearance of the tumor (Fig. 3), numerous postoperative laboratory procedures were carried out. Cephalin flocculation and thymol turbidity were within normal limits. Serum nonprotein nitrogen was 41 mg. per cent; cholesterol 200 mg. per cent; total serum protein 6.7 Gm. per cent with albumin 4.3 Gm. per cent and globulin, 2.4 Gm. per cent; amylase 233 mg. per cent reducing substance; phosphate 4.0 mg. per cent and alkaline phosphatase activity 2.0 Bodansky units. Bilirubin was 1.6 mg. per cent total, 0.9 mg. per cent direct. Bromsulfalein showed 2.0 mg. per cent retention. Intravenous urograms showed normal collecting system and ureters. Serologic tests for syphilis were negative. No evidence of generalized xanthomatosis could be detected. Liver and spleen were not palpable and no suggestive cutaneous lesions were present. Roentgenograms of the skull and long bones showed no abnormalities.

The patient was discharged asymptomatic on the thirteenth postoperative day. Physical examination of the chest at this time showed decrease in breath sounds at the right base with slight impairment of percussion. She has remained in good health in the nine months since discharge from the hospital.

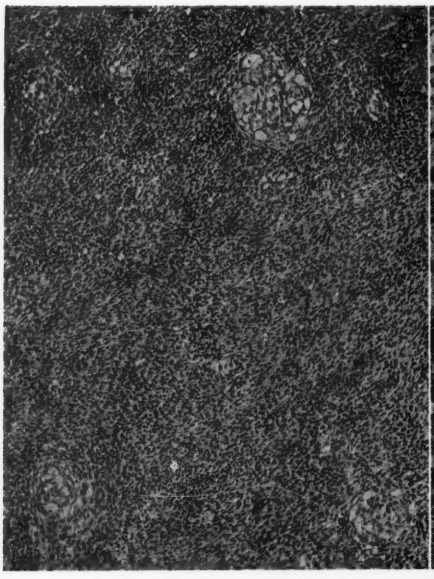

Fig. 4.

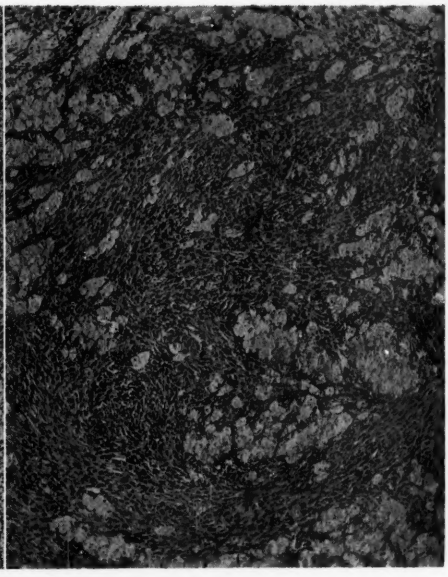

Fig. 5.

Fig. 4.—Luxuriant growth of oval or spindle-shaped cells with small groups of xanthoma cells.

Fig. 5.—Xanthoma cells scattered singly or in groups.

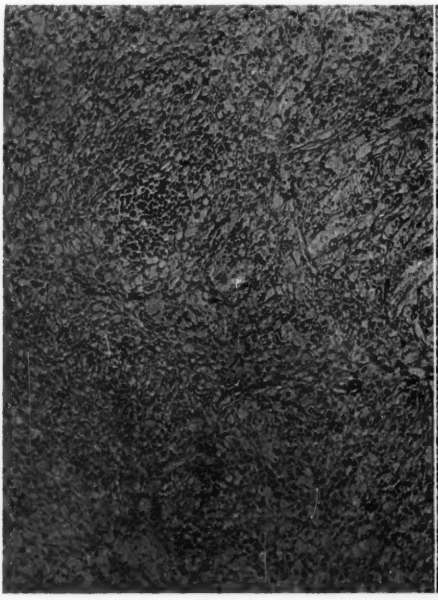

Fig. 6.

Fig. 7.

Fig. 6.—Area showing a preponderance of xanthoma cells. One giant cell and a small collection of lymphocytes are present.

Fig. 7.—Interlacing bands reminiscent of the structure of a neurinoma.

*Pathologic Study.**—

Gross description: The specimen consists of the lower lobe of a lung. The pleural surfaces are smooth and pink. The lung tissue is only slightly crepitant and within it a firm, round mass can be felt. On section this mass appears to be well circumscribed (Fig. 3), measures 4.5 cm. in greatest diameter and is composed of somewhat friable, yellow tissue, which has a smooth chalklike cut surface. In the center of the mass there is apparently a thin-walled blood vessel measuring about 1 cm. in greatest diameter and filled with blood clot. Small areas of hemorrhage are also seen around the periphery of the mass. The surrounding lung tissue is compressed and at one point only a thin layer about 2 mm. in thickness separates the tumor from the pleural surface. Representative blocks are taken for microscopic study.

Microscopic description: All of the sections show a luxuriant growth of oval or spindle-shaped cells (Fig. 4) lying on a background of eosinophilic fibrillary collagen. Scattered among them singly or in groups (Figs. 4, 5) are large, round, or polyhedral cells which have a small dark nucleus and foamy cytoplasm which stains deeply with sudan IV and is doubly refractile. Lymphocytes are scattered lightly throughout the sections and an occasional plasma cell and eosinophile are seen. A few multinucleated giant cells are also present (Fig. 6). In a few areas the collagenous material has a tendency to form interlacing bands reminiscent of the structure of a neurinoma (Fig. 7); no nervous elements can be demonstrated, however, either by the Masson trichrome stain or by the Wilder method. There are a few small areas of hemorrhage and foci of hemosiderin-laden macrophages are found scattered in the sections. The grossly described blood vessel in the center of the mass is seen to consist of an area of laked red blood cells, hemosiderin, and cholesterol clefts surrounded by hyaline scar tissue. Around the growth there is a definite connective tissue capsule which in some places is thin and partially broken through. Elastic stains do not reveal any lung framework within the mass itself.

Final Diagnosis.—Xanthoma in lung.

COMMENT

Considerable confusion exists concerning the nature and etiology of the disturbances of lipoid metabolism manifested by xanthomatous masses, whether local or generalized. Gruenfeld and Sielig[1] in 1934 and Thannhauser and Magendantz[2] in 1938 made exhaustive studies of the xanthomatous diseases. In general these authors consider the various forms of xanthoma to represent metabolic disturbances rather than neoplastic processes. They classify the xanthomatous diseases as (1) primary xanthomatosis with and without hypercholesterolemia, (2) secondary xanthomatosis associated with lipemia, and (3) localized xanthoma cell formation in true tumors.

In the case reported here there was no evidence of general metabolic disturbance as manifested by diabetes, hypercholesterolemia, or lipemia. No xanthomatous lesions were present in the skin and no osseous changes were demonstrable. Therefore, it would appear that the solitary tumor of the right lower lobe of the lung in our patient represents either a xanthomatous deposit of a primary nature or xanthomatous replacement of an underlying neoplasm, no identifiable remnants of which remain.

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*By T. P. B. Payne.

A REPORT OF A CASE OF LOCALIZED BRONCHOPULMONARY MONILIASIS SUCCESSFULLY TREATED BY SURGERY

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INTRODUCTION

BRONCHOPULMONARY moniliasis was first described by Castellani[1] in Ceylon in 1905. Many of his patients contracted the disease by inhaling tea dust in factories. In 1906 he found mycelial filaments and yeastlike bodies in the sputa of men suffering from tea tasters' cough. The clinical picture closely resembled tuberculosis, varying from mild cough to fever, hemoptysis, emaciation, and death. He struck a note of caution in making the diagnosis because monilia, which may exhibit all degrees of pathogenicity, are frequently found in the oral cavity of man. He described four pathogenic species: Monilia tropicalis Castellani, Monilia pinoyi Castellani, Monilia Krusei Castellani and Monilia metalondinensis Castellani, but indicated that other species with the same biochemical reactions may be nonpathogenic. He felt that pathogenic forms could be differentiated by intrapulmonary inoculation into rabbits with subsequent formation of characteristic pulmonary nodules.

In 1915 Boggs and Pincoffs[2] reported the first case of bronchopulmonary moniliasis in this country with post-mortem findings. Up until 1931 only five cases of bronchopulmonary moniliasis were confirmed by post-mortem examination. One of the most complete descriptions was made by Warr.[3] The disease may manifest itself in protean ways and affect the following structures: (1) tonsils, (2) bronchi, (3) conjunctiva, (4) intestinal tract, (5) ears, (6) urethra, (7) skin, (8) central nervous system. In addition, five instances of blood stream infection, all occurring in drug addicts, have been reported. Wessler and Browne[4] reported complete recovery of a patient with monilial bacteremia during the use of NaI intravenously and thymol and KI orally. Hiatt and Martin[5] reported cure of a patient treated with anti-candida rabbit serum, after iodides had been discontinued because of severe skin eruptions. Intravenous gentian violet has also been used. All forms of therapy have been only sporadically successful. Surgery has not been previously described as a therapeutic measure. The following case report represents the first successful surgical extirpation of localized bronchopulmonary moniliasis.

CASE REPORT

A 22-year-old, well-developed, white man was admitted to the hospital on Feb. 24, 1947, with a history of hemoptysis for three months. Three months before admission he developed

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a scanty cough with fever, and called a physician who confined him to bed for one week with "virus pneumonia." Because of continued cough with daily production of about one-half ounce of sputum, occasionally blood-streaked, roentgenograms of the chest were taken in November, 1946, and January, 1947. They revealed a persistent opacity in the upper lobe of the right lung and the physician made a tentative diagnosis of pulmonary tuberculosis. The patient had no dyspnea or easy fatigability and had gained two pounds in weight since the onset of the illness.

Physical examination revealed a well-nourished patient who did not appear ill. The temperature was 98.4° F. and the pulse rate was 76 beats per minute. Examination of the lungs was negative. X-ray views of the chest revealed an ovoid area of infiltration 3½ cm. in diameter in the axillary segment of the subapical division of the upper lobe bronchus (Fig. 1).

Laboratory studies revealed that the red cell count was 4,700,000, hemoglobin 81 per cent, and the white cell count 6,400 with a normal differential. Tuberculin intradermal test (PPD) was negative in dilutions of 1:10,000 and 1:1,000. Six concentrated specimens of the sputa and of the gastric juice were negative for tuberculosis and pathogenic fungi. A guinea pig inoculated with the sputum was autopsied in the eleventh week and tuberculosis could not be demonstrated. Coccidioidin skin test (1:1,000) was negative.

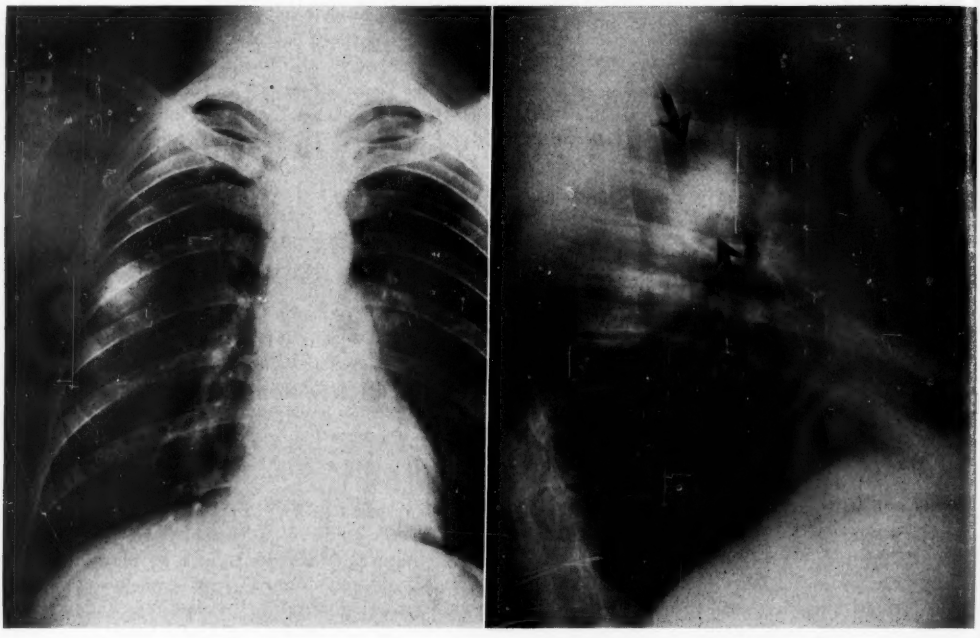
Sputum collected on May 13 and May 19 and cultured on Sabouraud's medium grew out numerous yeastlike colonies, which after subculture on sucrose, maltose, and lactose showed the cultural characteristics of *Candida albicans,* including branching forms on stab preparations.* On May 16, examination of the right chest was negative for fungi. On June 2, 1 c.c. of the culture of C. Albicans from the sputum was injected into a rabbit's ear vein. On June 10, autopsy of the animal revealed numerous bilateral renal granulomas, which were firm, yellow-white, and measured up to 1 cm. in diameter. Microscopically they consisted of acute granulomas in which neutrophilic and eosinophilic polymorphonuclear leucocytes predominated (Fig. 2). The surrounding tubules were filled with similar exudate.

Clinical Course.—The patient had an afebrile course but continued to raise about one-half ounce of sputum which was occasionally blood-streaked. Bronchoscopy and bronchograms were noncontributory. Because a lung abscess could not be ruled out, he was given a course of sulfadiazine (6 Gm. daily) and penicillin up to one million units daily. In addition, aerosol penicillin (30,000 units every four hours) was tried without any resultant diminution in the size of the lesion. Since carcinoma of the lung was a distinct possibility and since the lesion, if not malignant, might prove amenable to surgical extirpation, an operation was performed on May 2, 1947. The pleura was opened through the bed of the fourth rib. A circumscribed, somewhat firm lesion was observed in the axillary segment of the subapical division of the right upper lobe in close proximity to the oblique fissure. Since the lesion was firmly adherent to the parietal pleura, the latter was removed with the diseased portion of the lung. A wedge excision of the diseased axillary segment, which included the segmental bronchus, was performed. The edges of the defect were closed with short, running sutures of No. 00 catgut. A No. 20 Foley catheter was left in the pleural space and the wound was closed in layers with silk.

The patient made an uneventful postoperative recovery. On two occasions 350 c.c. of sanguineous, sterile fluid were removed from the pleural cavity. The patient was discharged on the nineteenth postoperative day. He was completely asymptomatic when seen one year later.

Pathology Report.—The specimen consisted of a 5.5 by 6 by 3 cm. wedge-shaped mass of lung tissue, to which was adherent a 4 by 1.5 by 0.7 cm. strip of muscle (Fig. 3). The lung was congested, red, and atelectatic. The base of the wedge consisted of both pleural layers, to which intercostal muscle was adherent. The apex of the specimen presented a 5 mm. channel with a 1 mm. thick, white wall, suggestive of a bronchus. Through this

*Because of the highly technical nature of fungus identification, a culture was submitted to Dr. Norman F. Conant of Duke University, who identified it as *Candida* (monilia) *albicans.*



A. *B.*

Fig. 1, *A* and *B*.—Anteroposterior and lateral x-ray views of the lungs reveal an ovoid area of infiltration in the axillary segment of the subapical division of the right upper lobe.

Fig. 2.—Low-power photomicrograph of rabbit's kidney, showing granuloma produced by intravenous injection of suspension of monilia grown from patient.

Fig. 3.—Lung specimen, showing bronchus at entrance to cavity, which is filled with semi-attached, gelatinous, monilial membrane.

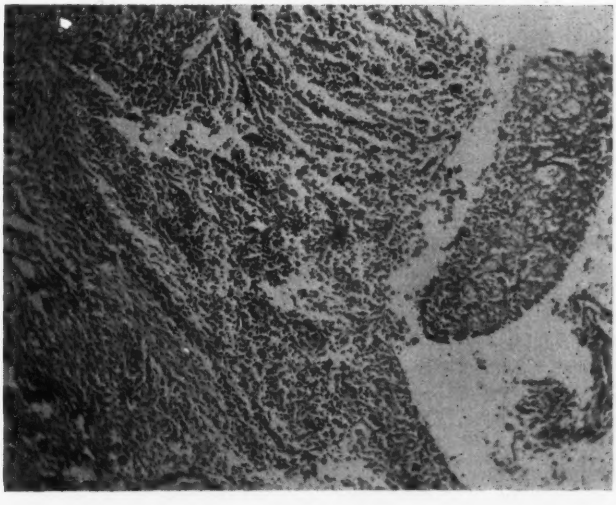


Fig. 4.—Low-power phototmicrograph of granulation tissue wall of lung cavity, showing loose mass of mycelia in the cavity.

opening projected a white, translucent, gelatinous, shiny membrane. The channel opened into a thick-walled, cystic structure measuring about 1.5 cm. in diameter and lined by the same translucent material, resembling a "thrush" membrane, which was removed, leaving a roughened, pebbly surface with numerous blood clots. The wall measured up to 1 cm. in thickness, was firm, and contained many small, yellow foci.

On microscopic examination (Figs. 4 and 5), the wall of the cavity was found to be lined by granulation tissue with numerous capillaries and fibroblasts and heavily infiltrated with plasma cells, and less heavily with lymphocytes, neutrophilic and eosinophilic polymorphonuclear leucocytes. The outer portion of the wall consisted of a layer of collagenous, fibrous tissue, in which eosinophiles and lymphocytes were the predominant white cells. The surrounding lung tissue was congested and atelectatic. About the blood vessels and small bronchi, lymphocytes formed follicles which showed considerable germinal activity. Adjacent to these follicles, the alveoli were frequently lined by cuboidal cells. Some of the alveoli and bronchi contained "dust cells" and vacuolated monocytes; others were filled with red blood cells and neutrophiles. In the area between the cavity and the pleura, the lymphoid accumulations were particularly dense. Only a few bronchi and channels lined by cuboidal cells

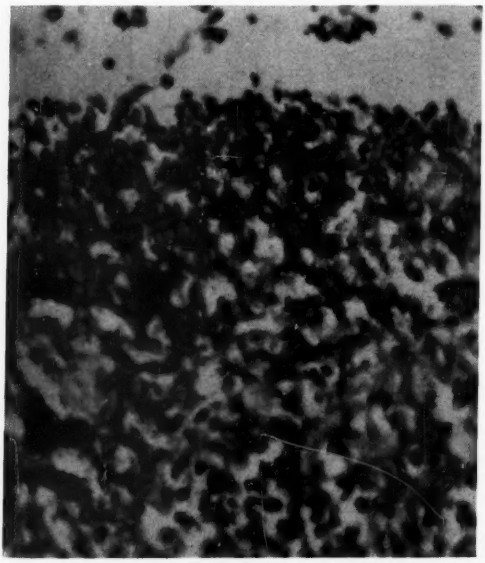


Fig. 5.—Oil immersion photomicrograph of mass of mycelia shown in Fig. 3.

remained. The channel at the apex of the specimen was lined by bronchial epithelium, which became ulcerated as it joined the cavity. The pleural layers and intercostal muscles were fused by vascular fibrous tissue, through which the blood vessels coursed in a radial direction. Lying loosely within the cavity there was a network of fungal mycelia, closely surrounded by neutrophiles. Frozen sections and hanging drop preparations of the loose, white membrane revealed that it was a growth of fungus, having the segmented, branching mycelia and chlamydospores characteristic of *C.* (monilia) *albicans.*

COMMENT

Bronchopulmonary moniliasis is a difficult diagnosis to make. Skin tests are equivocal.[6, 7] Monilia present in the sputum may be nonpathogenic or may be a secondary invader. Discovery of the characteristic

membrane of monilial infection in the diseased portion of the lung is the most valid diagnostic procedure. The appearance of a cavity connecting with a bronchus, filled with a gelatinous, white membrane and adherent to the chest wall, corresponds closely to the necropsy findings described by Warr.[3] Microscopic examination of the membrane proved the existence of numerous fungal elements, identifiable as C. (monilia) *albicans*. Attempts to demonstrate other agents by bacteriologic and histologic methods failed. There was also failure to elicit a history of any antecedent disease. Further evidence of the association of monilia with the pulmonary lesion was the consistent appearance of the typical organisms in the sputum. These organisms from the sputum injected intravenously into a rabbit's ear[8] produced granulomas of the kidneys resembling the nodules in the lungs of a rabbit produced after intrapulmonary inoculation by Castellani.[1] Clinically, the sputum was scanty. The glairy, sticky quality with a sweet or yeastlike odor described by others was not present. The gross and microscopic picture of the lesion, the demonstration of monilia in the membrane lining the cavity, the finding of monilia in the sputum by direct examination and culture, a positive virulence test in an inoculated rabbit, and the failure to demonstrate any other causative agent or to elicit a history of antecedent disease are the reasons for calling this a case of primary bronchopulmonary moniliasis. Only the lung was invaded in this case.

Vaccine and drug therapy have been only sporadically successful.[1, 4, 5, 9, 10] Surgical intervention should be considered as an effective therapeutic procedure in the treatment of localized bronchopulmonary moniliasis.

SUMMARY

The first case of primary bronchopulmonary moniliasis successfully treated by surgical excision has been reported. The patient has shown no clinical or x-ray evidence of recurrence of the disease during the year following operation.

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CONSTRICTIVE PERICARDITIS WITH OBSTRUCTION OF PULMONARY VEINS

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SINCE Chevers'[1] excellent description of chronic pericardial disease in 1842, there have been many publications about this disease. Most authors separate the disease into constrictive pericarditis, a fibrous process of the pericardium without external adhesions, and chronic mediastinopericarditis, a process in which the external adhesions are predominant. In some cases such a clear-cut distinction cannot be made.

Effective surgical therapy for constrictive pericarditis had not been accomplished until three decades ago. Brauer[2] in 1902 described a rib removal procedure called cardiolysis. This, however, was of value only in the mediastinopericarditis group despite its indiscriminate usage. Delorme[3] in 1898 proposed removal of the constricting pericardial sac, but reports of pericardial resection did not appear until 1920. In the ensuing ten years there were reports of surgical cures by Rehn,[4] Volhard and Schmieden,[5] Sauerbruch,[6] Churchill,[7] and Beck.[8] In the last two decades there has been more widespread use of this procedure.

In a typical case of constrictive pericarditis there are symptoms and signs of right-sided heart failure in a patient with a small quiet heart. The pericardial scar limits diastolic filling, thereby decreasing stroke volume. In most cases this has a marked effect on the systemic venous circulation, producing persistent serous effusions, edema, and elevated venous pressure. There is also tachycardia, low pulse pressure, and pulsus paradoxus. Detailed physiologic, clinical, and surgical discussions of this condition and its variations have been published by Beck and Griswold,[9] White,[10] Heuer and Stewart,[11] Blalock and Burwell,[12] and Sellors.[13]

Different classifications and concepts of pathogenesis have been expressed through the years by Pick,[14] Kelly,[15] Rolleston,[16] Curschmann,[17] Rothstein,[18] and others. Constrictive pericarditis has been described under other names such as Pick's disease, adhesive pericarditis, and polyserositis. Much of this variation is probably due to the unknown etiology of the majority of cases reported.

Most writers report approximately 20 per cent of their cases as proved tuberculosis (Cole and associates[19] and Harrison and White[20]). In contrast, Blalock and Burwell[12] reported the tubercle bacillus as etiologic agent in 64 per cent of their twenty-eight surgical cases. Another group of cases are regarded as sequelae of pyogenic pericarditis. Rheumatic pericarditis is now

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considered a rare cause of constrictive pericarditis although it is responsible for most asymptomatic adhesive pericardiums and mediastinopericarditis. (White,[10] Harrison and White,[20] Sprague and co-workers,[21] Sellors,[13] and others.)

With few exceptions the remaining cases are classified as idiopathic. All but one of our eight cases fell into this category. It was said by Trout[22] that the problem in establishing the etiology in this group is due to the fact that an organism causing a prolonged defensive process would be difficult to find late in the disease. Some authors feel that these are unproved cases of tuberculosis. Wells[23] postulated a spread of sclerogenic products of tubercle bacilli to pericardial and pleural surfaces from adjacent tuberculous lymph nodes. Due to lack of evidence, the etiology in a large proportion of cases remains undetermined.

The following case is being reported because of an unusual basis for unsuccessful surgical therapy. The pulmonary veins were markedly stenosed by fibrous tissue. This unique phase of the process was not disclosed until autopsy.

CASE REPORT

A white boy, (No. 376848), aged 14 years, was seen for the first time at the University of Chicago Clinics on Feb. 27, 1946. Complaints were swelling of the abdomen for five to six weeks, dyspnea for four weeks, and ankle swelling for several days. Past history revealed an episode of severe dyspnea three years previously which subsided in three days. A chest roentgenogram at that time by a local physician showed a marked semicircular bulge on the left heart border in the region of the pulmonary conus. He was asymptomatic until October, 1945, when he was admitted to a contagious disease hospital for a short period with a questionable diagnosis of meningitis. In January, 1946, he developed a respiratory infection from which he recovered in two weeks. Two weeks subsequently, however, he noticed an abdominal enlargement. Paracentesis yielded four quarts of straw colored transudate from which nothing was cultured. In the remaining three weeks before admission to the hospital he required two more paracenteses.

This boy had had the common childhood illnesses and had not had rheumatic fever. The family history was noncontributory.

Physical examination revealed a well-developed, but not too well-nourished, boy. There was bilateral pleural effusion and râles at the left base. The respiratory rate was 24 per minute. The heart was enlarged to percussion. On auscultation the heart sounds were distant and indistinct. No friction rub nor murmurs were heard. The heart rate was 130 and there was a definite pulsus paradoxus. The blood pressure was 112/88. The abdomen was very distended and tense, and a fluid wave was present. No organs were palpable, but the liver was percussible two fingers below the costal margin. There was 1 plus edema of the ankles.

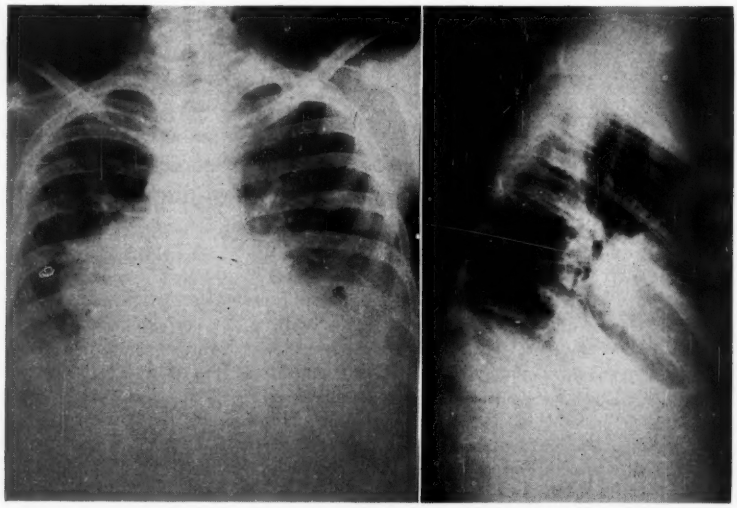
Laboratory.—Blood examination revealed a red blood cell count of 5.29 million, hemoglobin 15 Gm., hematocrit 48, and white blood cell count 7,050. The differential count was 62 per cent polymorphonuclears, 34 per cent lymphocytes, 3 per cent monocytes, and 1 per cent eosinophiles. Wassermann, Kahn, Mantoux in 1:1000 concentration, and the tuberculin patch test were all negative. The sedimentation rate was 5 mm. per hour (Wintrobe). Blood chemistry values were: nonprotein nitrogen 22 mg. per cent, plasma proteins 6.5 Gm. per cent, albumin 3.14 Gm. per cent, globulin 3.29 Gm. per cent, albumin-globulin ratio of .95; serum cholesterol 136 mg. per cent, and alkaline phosphatase 8.6 units. The urine was normal.

X-ray views of the chest showed bilateral pleural effusion and a large rounded mediastinal mass on the left side. The heart was enlarged. Aneurysm of the aorta or pulmonary artery

and mediastinal tumor were considered possible diagnoses from the x-ray appearance (see Fig. 1, *A* and *B*).

Special Tests.—The venous pressure in the right antecubital vein was 319 mm. and 320 mm. of water on two measurements. Vital capacity was 1.5 liters. The electrocardiogram was interpreted as showing right axis deviation. There was low voltage of the ventricular complexes.

Working Diagnosis.—A working diagnosis was made of tumor or cyst in mediastinum or pericardium with impingement upon or obstruction of the pulmonary circulation.



A. *B.*

Fig. 1.—*A*, Chest roentgenogram of patient at time of admission. Note bilateral pleural effusion and sharply defined capacity extending out from left border of heart. *B*, Lateral chest x-ray view showing the capacity to be in the anterior mediastinum.

Preoperative Course.—The boy was observed seven weeks before surgical exploration. Attention was directed to the generalized fluid accumulation. Two days after admission (March 2, 1946) a paracentesis was performed yielding 4,200 c.c. of bright orange-yellow fluid with a specific gravity of 1,016. Total protein determination gave a value of 3.20 Gm. per cent. Tests for bile were negative. Microscopically, red blood cells were seen. Nothing grew on culture of the fluid.

Three days later (March 5, 1946) a paracentesis yielded 700 c.c. of bloody fluid with a specific gravity of 1.010, total protein 1.08 Gm. per cent, albumin .55 Gm. per cent, globulin .53 Gm. per cent, and nonprotein nitrogen 22 mg. per cent. The patient had decreased in weight from 69.4 to 60.8 kilograms in this one week of preoperative preparation.

Operation.—The anesthesia was ethylene-oxygen plus ether under positive pressure. High oxygen concentration was necessary throughout the procedure.

An incision was made encircling the lower aspect of the left breast, and the left pleural cavity was entered through the left fifth interspace after division of the fourth and fifth costal cartilages. Considerable pleural fluid escaped as the cavity was opened. The left lung was found to be very firmly fixed to the heart and mediastinum anteroinferiorly and both visceral and parietal pleura in the region were markedly thickened. A firm tumor mass approximately 2 to 2½ inches in diameter was found located in the anterior mediastinum directly over the pulmonary conus. It was bound down firmly with dense adhesions due to thickening of the mediastinal pleura.

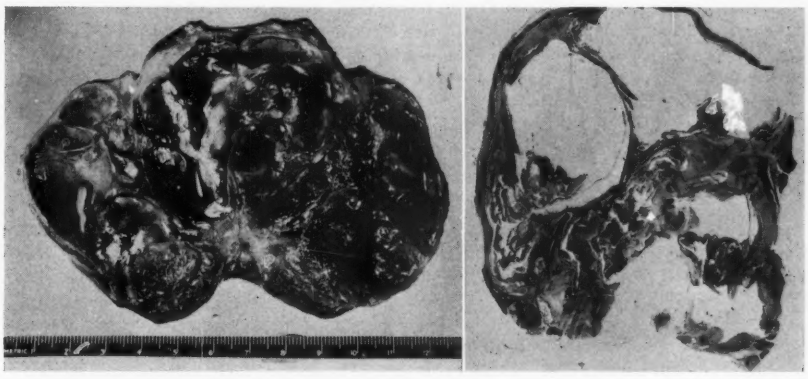
After mobilizing the lung from the pericardium and mediastinal pleura, a cleavage plane was found between the tumor mass and the pericardium, and by blunt and sharp dissection it was gradually freed from these structures. It was firmly attached to the pericardium by dense fibrous adhesions. No large blood vessels were encountered.

As soon as the mass was mobilized the conus showed considerable pulsation, and it was evident that this had been considerably obstructed by the mass. On release of this obstruction, the patient's respiratory rate dropped from 42 to 28, his color improved perceptibly, and the pulse rate as well as quality improved. The mass was completely removed.

The chest wound was closed in layers, a pezzar suction catheter being brought out through a stab wound for drainage.

The patient was given 1,150 c.c. of whole blood and 300 c.c. of normal saline solution while on the operating table.

The mass was a multiloculated cyst about 8 by 6 by 5 cm. with walls varying from 1 to 5 mm. The cyst contained some clots and some reddish brown fluid which, analyzed chemically, gave these values: total protein 5.75 Gm. per cent, albumin 2.75 Gm. per cent, globulin 3.0 Gm. per cent, nonprotein nitrogen 41.3 mg. per cent, sugar 34 mg. per cent, total cholesterol 265 mg. per cent, and free cholesterol 179 mg. per cent (see Fig. 2, *A*). Histologically it consisted of fibrous walls and organizing granulation tissue as a cyst lining. There were hemosiderin deposits and a few lymphocytes and plasma cells scattered throughout the connective tissue. Several large endothelial-lined vascular structures filled with blood elements established the pathologic diagnosis as cavernous hemangioma of the mediastinum (See Fig. 2, *B*).



A. *B.*

Fig. 2.—*A*, Photograph of opened tumor mass removed from over the pulmonary conus in the anterior mediastinum. Note multiloculation with considerable fibrosis in the trabeculae. *B*, Low-power microscopic section through entire tumor mass. Note spaces of various sizes and shapes partly filled with blood. Some areas of calcification may be seen.

Postoperative Course.—The patient did well following operation and appeared to be improved, but only for a short time. The wound healed by primary intention. Fluid continued to accumulate in the peritoneal and pleural cavities, however, and the venous pressure remained elevated. The left pleural cavity gradually became obliterated. Paracentesis and thoracentesis on the right side of the chest were carried out and diuretics given.

On fluoroscopic examination the cardiac movements were markedly diminished. This finding, along with increased venous pressure, strongly suggested the diagnosis of pericardial effusion or constrictive pericarditis. Aspiration of the pericardial cavity revealed a relatively small amount of bloody fluid. An exploration was thought to be indicated. Plasma proteins at this time were 7 Gm. per cent, albumin 3.1 Gm. per cent, and globulin 4 Gm. per cent.

Operation.—The operation was performed on April 4, 1946. The incision extended from the left third costal cartilage, downward over the left border of the sternum, and outward over the sixth costal cartilage. The pectoralis major muscle was reflected laterally and the fourth, fifth, and sixth costal cartilages were removed subperichondrially along with a short stump of the anterior ends of the fourth and fifth ribs. The left pleura and lung were then dissected laterally from over the perichondrium after ligation of the left internal mammary vessels at the upper and lower end of the exposed area.

The pericardium was definitely thickened and very little cardiac movement could be seen. On opening the pericardium, hemorrhagic transudate spurted out under high pressure. The incision in the pericardium was enlarged and a large amount of fluid was removed. The anesthetist stated that the color of the patient improved immediately and the blood pressure increased to 130/100. The pericardium was then excised over an area approximately 2½ by 2 inches and a large Penrose drain was placed on the bottom of the pericardial cavity.

Exploration with a finger revealed no adhesions between the pericardium and the heart muscle. The epicardium was somewhat thickened but did not interfere with heart activity. The chest wound was then closed in layers. The Penrose drain was sutured to the skin. During the procedure the patient received 200 c.c. of whole blood and 200 c.c. of normal saline solution.

The thickened pericardium removed at surgery showed perivascular infiltration of chronic inflammatory cells. Nothing was cultured from the fluid. A guinea pig into which the fluid was injected was later sacrificed and found to be normal.

Postoperative Course. The patient made a satisfactory recovery with routine management. The wound healed readily. The cardiorespiratory function improved considerably. With the occasional aid of diuretics, the pleural fluid and ascites markedly decreased and the ankle edema disappeared. The boy felt well and was able to walk about in his room.

On May 3, 1946, a grayish-white membrane on the palate was observed, considered Vincent's angina, and treated as such. One week later the diagnosis of moniliasis was established. The lesions involved the pharynx, nasopharynx, larynx, and trachea by that time. They were treated locally with 2 per cent aqueous gentian violet and systemically with sulfadiazine, 5 Gm., and sodium bicarbonate, 3 Gm. After gross hematuria on May 16, 1946, the systemic therapy was abandoned. The visible lesions disappeared after two weeks of therapy, but therapy was continued for two months.

After May 10 his condition in relation to the primary disease was relatively stationary. Urinary output averaged around 600 c.c. per day except for occasional days when 1 c.c. of Mercupurin was given to induce diuresis. Breathing was difficult at times, but pleural fluid by x-ray and fluoroscopy was decreasing in amount. Some ankle edema and ascites accumulated in mid-June and paracentesis was done June 18. The 4,870 c.c. of clear yellow fluid had a specific gravity of 1.020.

An x-ray on June 27 showed the heart to be relatively normal in size and a small amount of pleural fluid was present (see Fig. 3).

Ascites and ankle edema became evident again on July 10, 1946. There was venous engorgement and the venous pressure was 300 mm. of water.

Mercupurin (1 c.c.) was given to promote a diuresis about once a week and the patient continued to pursue a somewhat static course. Plasma proteins, nonprotein nitrogen, and the venous pressure remained at the previous levels.

Another paracentesis of 3,400 c.c. was done July 29. Despite removal of large quantities of pleural and ascitic fluid the plasma proteins were at no time subnormal. The albumin-globulin ratio, however, was always less than one.

By August the disease process was thought to have entered a chronic phase and further surgery seemed indicated.

Operation.—Aug. 13, 1946, partial pericardectomy was done. An incision was made through the scar of the former operation, the upper and lower angles being extended.

The soft structures of the chest wall were reflected to the left from over the pericardium. Parts of the third, fourth, and fifth ribs were removed subperiosteally to allow exposure. The left pleura and lung were then reflected from over the pericardium, and the former opening into the pericardial sac was found to be closed by fibrous tissue.

Further exploration revealed the pericardial sac to be completely obliterated. After rongeuring away a considerable amount of the lower sternum, the pericardium was freed from the heart, first over the left ventricle anteriorly and to the left, and then over the right ventricle anteriorly. Two pieces of pericardium, measuring about 3 by 1½ inches each, were removed. The heart enlarged noticeably as the pericardium was freed from its surface.

The soft structures of the chest wall were then sutured over the heart after placing one Penrose drain in the wound.

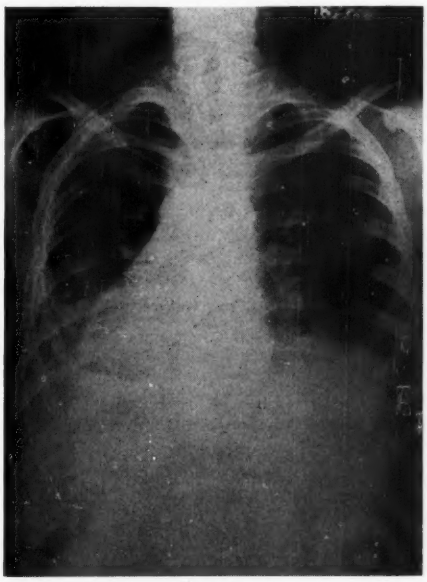


Fig. 3.

Fig. 4.

Fig. 3.—Chest roentgenogram taken almost four months after removal of mediastinal hemangioma and three months after partial pericardectomy. Note improved aeration of the lungs due to decrease in pleural effusion.

Fig. 4.—Chest x-ray view one day prior to expiration; there is considerable bilateral pulmonary congestion.

Postoperative Course.—The patient received the routine care including penicillin and nasal oxygen. Soon after surgery he was given 300 c.c. of whole blood, the transfusion being terminated because the venous pressure was found to be 375 mm. of water.

On the second postoperative day the temperature was 37°C., pulse rate 110-120, and respiratory rate 30 per minute. Blood pressure was 100/65. The hemoglobin was 10.5 Gm. and white blood cell count 5,000. The urinary output after surgery was only 300 c.c. per day.

Blood chemistry on the third postoperative day revealed a slight alkalosis; the carbon dioxide capacity was 75 vol. per cent, pH 7.45, and chlorides 85 mM/1. Plasma proteins were 6.62 Gm. per cent, albumin 3.19 Gm. per cent, globulin 3.45 Gm. per cent, albumin-globulin .93, and nonprotein nitrogen 52.6 mg. per cent. Ammonium chloride, 3 Gm., was started and the following day the patient received 300 c.c. of whole blood and 60 c.c. of normal saline intravenously.

There was a slight increase in urinary output and on Aug, 19, 1946, the sixth postoperative day, the carbon dioxide capacity of the plasma was 66.8 vol. per cent and the pH was 7.4. Plasma proteins and nonprotein nitrogen were only slightly altered from the values on Aug. 16, 1946. The sedimentation rate, as on every determination throughout the course, was not elevated.

A paracentesis on Aug. 18 yielded 2,950 c.c. On Aug. 22 an x-ray examination of the chest revealed bilateral congestion of the lungs (see Fig. 4).

Respiration became labored on Aug. 20, one week after surgery, and the urinary output was only 200 c.c. per day. On Aug. 22 the boy became cyanotic and respiration was still more difficult. A chest film showed passive congestion and a small amount of pleural fluid. The output that day was only 150 c.c.

At 4 A.M. on Aug. 23 his condition became decidedly worse. He was very cyanotic, respirations were slow, pulse was very weak, and he could not be aroused.

He improved slightly in the early morning with repeated doses of caffeine and sodium benzoate, Coramine, and Digalen. However, respiration became very weak, pulse irregular, and the extremities cold and moist. Râles and ronchi were heard in the chest. He died at 12:40 P.M.

Autopsy.—The body showed emaciation and a dilation of venous collaterals on the trunk. There was chronic polyserositis with generalized edema and chronic passive congestion.

There was obliterative fibrous pleuritis and pericarditis, except for the anterior pericardium which had been surgically removed. The pulmonary veins were very stenosed by rigid fibrous rings where they passed the pleuropericardium. The inferior pulmonary veins would not admit a finger tip and the superior vessels would barely do so. The superior and inferior vena cavae were distended but were not stenosed at any point. The aorta and pulmonary artery were normal.

The heart was distorted in shape due to a definite right ventricular hypertrophy. The valves were all normal.

The lungs demonstrated an extreme degree of chronic passive congestion and had areas of bronchopneumonia and chronic interstitial pneumonitis. A small peribronchial lymph node had an encapsulated (inactive) area of tuberculosis.

There were 2,100 c.c. of ascites. The peritoneal surfaces were irregularly thickened and white. There were many adhesions between the bowel and abdominal organs.

In addition to adhesions, the capsule of the liver was everywhere thickened. An extreme degree of fibrosis in the region of porta hepatis stenosed the portal vein in this area. Distal to the stenosis the intima of the vein was thickened.

The bowel wall was increased in thickness by 50 per cent due to edema and fibrous white thickening of the serosa. The mesentery was short and stiff and there were many adhesions between adjacent loops of the bowel. They tended to form cystlike structures.

The kidneys, pancreas, liver, and spleen all demonstrated chronic passive congestion. The kidneys on microscopic examination had many hyaline and granular casts, and many round bodies lodged in the tubules. This was considered obstructive nephropathy due to sulfa drug.

Histologically, the over-all picture was one of much vascular fibrosis on all serous surfaces with minimal amounts of inflammation.

DISCUSSION

In this case a congenital (cavernous hemangioma) and inflammatory lesion of the mediastinum were coexistent. The effect of the pleuritis and pericarditis on the circulation was enhanced by the congenital lesion compressing the pulmonary conus. The simultaneous action of these two processes was shown by the fact that the left pleural cavity was partially obliterated at the time of surgical removal of the hemangioma and the pathologic specimen demonstrated some chronic inflammation.

After the congenital lesions was removed there was temporary improvement in the patient's condition. However, progression of the inflammatory process and accumulation of pericardial fluid required further surgical intervention. Drainage of pericardial fluid at the second operation again led to marked improvement which was of several months' duration. This was terminated by the steady increase in constriction produced by the process in the pericardium. Surgical decortication of the compressed heart allowed the heart to increase diastolic filling. The pulmonary vein constriction impaired the pulmonary circulation to such an extent, however, that improvement did not take place. One week after surgery the patient began a rapid downhill course, terminating a few days later.

In the typical case of constrictive pericarditis the fibrous scar firmly encases the heart and there is no cardiac enlargement. Sellors[13] said that the pulmonary circulation, in contrast to the systemic, is relatively unaffected in this condition because a small volume is pumped into it. The right ventricle in this patient was greatly hypertrophied, however, and this was probably due to mechanical stress placed on the pulmonary circulation. This can be attributed to (1) the tumor compressing the pulmonary conus, and (2) the constriction of the pulmonary veins. The latter was of such severity that release of the heart from its constricting pericardium was not sufficient for recovery of normal circulatory mechanism.

In a search through the literature on constrictive pericarditis we have been unable to find a case of pulmonary vein constriction proved by post-mortem examination. There are many cases reported in which it has been proved that the fibrous process had involved the superior and/or the inferior vena cava. Gouley[24] reported six cases of chronic mediastinopericarditis with pulmonary artery constriction proved by post-mortem examination. Egan[25] in a pathologic report described constriction of the pulmonary vein orifices by calcium rings. This, however, was in a case of calcium deposition in the cardiac musculature.

At post-mortem there was an extensive fibrous peritonitis. The portal veins was markedly compressed by fibrous adhesions at the porta hepatis. Rolleston[16] said this occurrence was very rare even when the portal fissure was involved by perihepatitis. White[26] also said that this must be very rare because jaundice was so rare in these cases. He assumed that simultaneous constriction of the common bile duct must occur.

There was an unusual opportunity in this case to study the development of constrictive pericarditis from the onset of the process. Before many changes had occurred in the pericardium, symptoms brought the patient to the clinic. Probably the additional effect of the congenital lesion was instrumental in this respect.

RESULTS IN ADDITIONAL CASES

At the University of Chicago Clinics seven additional patients with constrictive pericarditis had surgical decortication of the heart. They ranged in age from 14 to 31 years, except for one man of 47 years. Etiology could only be determined in one patient and this was tuberculosis.

Six of the patients were sufficiently improved to carry on normal life activities. Of these, two required a second pericardial resection to attain this result. Complete recovery in two of this group was not achieved until digitalization was carried out.

In addition to the case reported in this paper there was one other death. This occurred in the patient of 47 years who had active tuberculosis as the cause of chronic pericarditis. There was no clinical improvement after surgery and the patient died seven months later. Post-mortem examination was not performed.

There was evidence of great vessel involvement only in the case which is the subject of this report. We consider the severe constriction of the pulmonary veins the basis for failure of surgical therapy in this patient.

SUMMARY

A case of constrictive pericarditis is presented in which the patient was studied from the onset of the process to its fatal termination. Surgical therapy was ineffective due to a unique process not disclosed until autopsy, namely, severe stenosis of the pulmonary veins by fibrosis in the pleura and pericardium. Decortication of the heart ordinarily yields a high percentage of cures in these patients as demonstrated by the results in seven additional cases.

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PRIMARY LYMPHOSARCOMA OF LUNG

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PULMONARY lesions are common in lymphomatoid diseases, but the literature contains very few reports of primary lymphosarcoma of the lung. In an analysis of 196 cases of lymphosarcoma proved by biopsy, Sugarbaker and Craver[1] found that the lung was the apparent primary site of lymphosarcoma in only one instance, or 0.5 per cent of the cases. A case is herewith reported in which a large lymphosarcoma involving about one-half of the left lung was removed by pneumonectomy. The patient was in excellent general condition and free of evidence of disease for over one and one-half years following operation. Then roentgenograms revealed a small lesion in the other lung which was successfully treated by radiation therapy. Three years have now elapsed since the pneumonectomy and the patient appears to be well at this time.

CASE REPORT

M. M., was a man aged 50 years. Three months prior to admission the patient was examined because of a slight exacerbation of symptoms caused by a chronic gastric ulcer. On fluoroscopic examination a lesion was discovered in the left lung. The patient had no pulmonary symptoms. There was no cough, chest pain, hemoptysis, dyspnea, or loss of weight. On physical examination the patient appeared well developed and somewhat obese. He did not appear ill. There was no evidence of lymphadenopathy. The positive findings on physical examination were dullness over the upper half of the left lung field anteriorly and over the upper third posteriorly. Bronchial breath sounds were present over the same area. There were occasional crepitant râles at the end of inspiration. The remainder of the left lung field and the entire right lung field were clear. A roentgenogram of the chest showed asymmetry of the thorax with narrowing of the interspaces on the left side. The left leaf of the diaphragm was elevated but not paralyzed. The heart and mediastinal structures were slightly displaced to the left. The area of the left upper lobe was uniformly dense (Fig. 1). A bronchoscopy showed no evidence of an endobronchial lesion, and no distortion or fixation of the tracheobronchial tree. Exploratory thoracotomy was recommended. The diagnosis was in doubt. Because of the large size of the tumor mass and the complete absence of pulmonary symptoms, a bronchiogenic carcinoma, although still a definite possibility, did not seem likely. On Oct. 23, 1944, an operation was performed and the findings were as follows.

The left upper lobe was diffusely adherent to the chest wall by adhesions which were moderately vascular but readily divided. The entire upper lobe was dense, but did not have the firm consistency usually found with carcinoma. The surface of the upper lobe was gray in color. The lower lobe was air-containing. The main bronchus showed no abnormality at the site of section. The mediastinal and bronchial lymph nodes were not enlarged. A hilar dissection type of total left pneumonectomy was performed. The postoperative course was uneventful.

Pathologic examination of the lung revealed almost the entire upper lobe to be replaced by a tumor mass of relatively soft consistency. The lower lobe was aerated and appeared relatively normal. On cut section the upper lobe was occupied by a tumor which was

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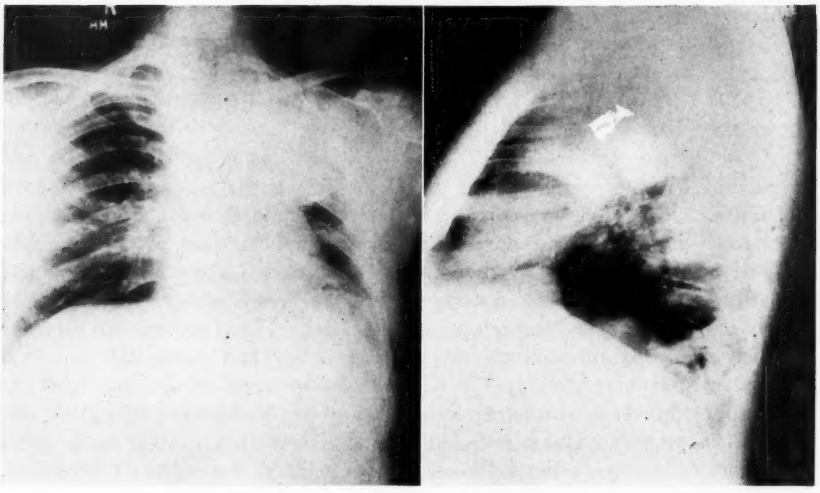
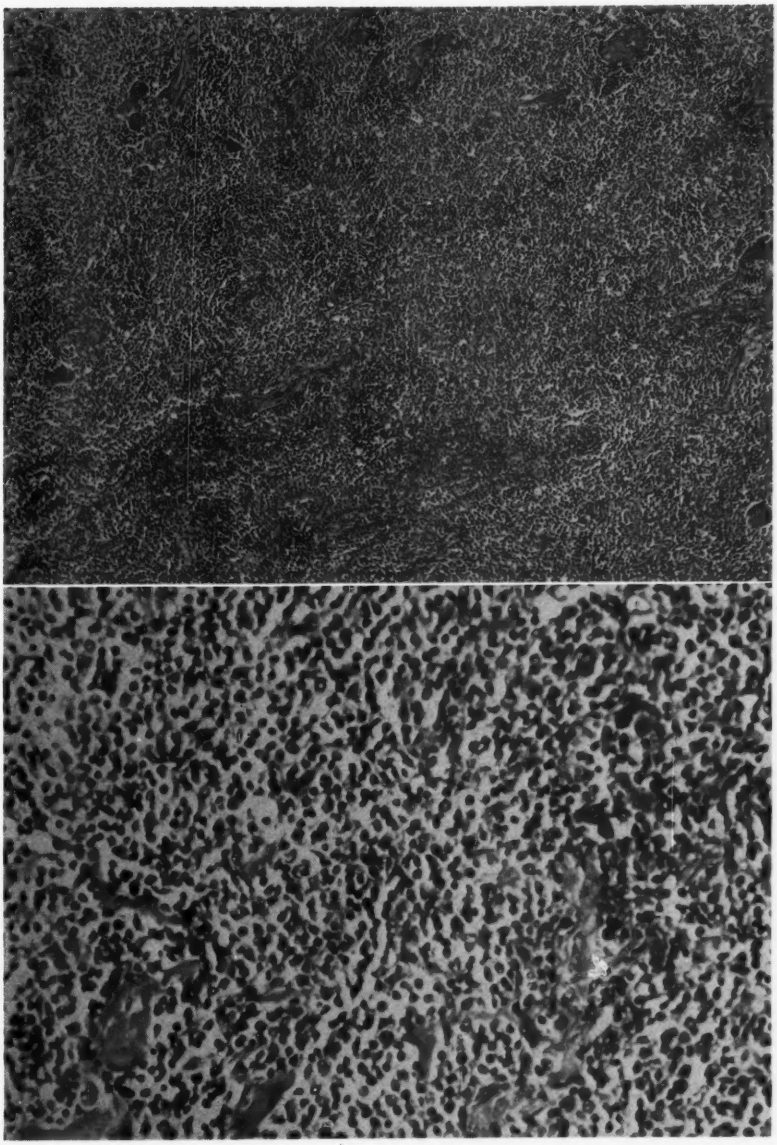


A. *B.*

Fig. 1.—*A* and *B*, Roentgenograms show density in upper half of left lung field.

Fig. 2.—Cut section of left lung. Tumor is lighter in color than normal lung. Note how well the bronchi are preserved and stand out on the cut surface.

A.



B.

Fig. 3.—*A* and *B*, Photomicrographs of tumor; note the uniform appearance of the lymphoblasts.

pale gray in color and which thickened the lung tissue uniformly without causing it to lose all of its elasticity. All the bronchi were preserved and stood out on the cut surface of the lung (Fig. 2). The tumor crossed the interlobar septum, obliterating it, and invaded the lower lobe for a short distance. The remainder of the lower lobe seemed normal. The pleura was thickened where the tumor came in contact with it, but there was no extension of the tumor through the pleura. The main bronchi were free of tumor.

On microscopic examination the lung was extensively infiltrated by a tumor composed largely of lymphoblasts mixed in places with an occasional reticulum cell (Fig. 3). The pulmonary substance had been largely replaced not only by the infiltrative growth but by an extensive process of fibrosis. Mitoses were rare and relatively difficult to find. The lesion appeared to be a solitary and apparently primary lymphosarcoma of the lung which must have grown slowly and in a relatively restrained fashion, since there was a good deal of associated fibrosis. There was no evidence of lymph node involvement.

The patient continued to be asymptomatic and remained in excellent health. Roentgenograms of the chest were taken at suitable intervals. In May, 1946, nineteen months after operation, a small area of density was noted in the outer portion of the right midlung field. The lesion increased in size very slowly during the following seven months. The patient remained completely asymptomatic. In December, 1946, radiotherapy was instituted.* A total of 1,150 r was given to each of two fields over the right side of the chest with almost complete disappearance of the lesion on the roentgenogram. In July, 1947, only a linear density remained on the film. The patient has gained ten pounds during the past three years. There has never been any evidence of lymphadenopathy.

All types of primary sarcoma of the lung are rare. In past years small round-cell or oat-cell carcinoma of the lung has been confused with sarcoma. The gross and microscopic appearance of the tumor described here differs from the small-cell carcinoma. Lymphosarcoma arising in the lung has been considered to be so unusual that its existence has even been doubted by some pathologists. In contrast, however, is the frequent occurrence of involvement of the lung secondary to lymphosarcoma of the bronchial and mediastinal lymph nodes. Vieta and Craver[2] analyzed 239 cases of lymphosarcoma in which roentgen studies of the lungs were made. Infiltration of the lung occurred in 23.8 per cent. The parenchymal infiltrations as shown on the roentgenogram were usually diffuse, but in 5.8 per cent isolated nodular masses occurred. In many instances the infiltration appeared to effect the greater portion of the lobe of the lung by direct extension from the hilar structures. Formation of discrete, lobular foci was nearly always accompanied by infiltrative lesions.

Churchill[3] recently reported a case of primary lymphosarcoma of the lung successfully treated by lobectomy. This 45-year-old woman complained of bouts of fever and generalized malaise. She had experienced vague pains in the right thoracic region for many years. The roentgenogram showed a large dense area in the right upper lobe which did not correspond with a bronchopulmonary segmental distribution. There was a slight reduction in the volume of the diseased portion of the lung. The lymph nodes were apparently uninvolved. The patient was given radiation therapy postoperatively and was well six months later.

Spatt and Grayzel[4] reported a case of primary lymphosarcoma of the lung which was also successfully treated by lobectomy. Their patient was a 34-year-old woman who complained of moderate cough and bloody sputum. Roentgenograms showed a small egg-shaped mass in the right lower lobe. The surgical

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specimen revealed a moderate-sized bronchus extending into the mass and fusing with it. Small bronchiectatic cavities were also present. Microscopic examination revealed the tumor cells to be particularly prominent about the bronchi and bronchioles and to a lesser degree about the blood vessels. The patient was well fourteen months after operation.

In most cases of lymphosarcoma the involvement is too diffuse to permit complete surgical removal of the diseased tissue, and radiation therapy is the treatment of choice. When the process is sufficiently localized, radical surgical excision, perhaps combined with radiation therapy, may give the best results. Lymphosarcoma usually involves the lung secondarily, but when the pulmonary lesion is apparently primary, lobectomy or pneumonectomy may be indicated. No conclusions as to the relative merits of surgery and radiation therapy in the treatment of primary lymphosarcoma of the lung can be drawn from my case. Since the lesion which later appeared in the right lung responded well to radiation therapy, it is quite possible that a favorable result might also have been obtained if the original large lesion in the left lung had been irradiated. A positive diagnosis of primary lymphosarcoma would be difficult to establish, however, unless an exploratory thoracotomy were performed.

SUMMARY

A case of a large, apparently primary lymphosarcoma of the lung treated by pneumonectomy is reported. A small lesion which later appeared in the opposite lung responded well to radiation therapy. The patient never had any pulmonary or other symptoms referable to the lymphosarcoma, and is clinically well over three years after pneumonectomy. Two previously reported surgically treated cases of primary lymphosarcoma of the lung without mediastinal lymph node involvement are briefly reviewed.

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COMPLETE DISRUPTION OF THE ESOPHAGUS

Report of Case

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IT IS interesting to note that the earliest recorded case of rupture of the esophagus was one of complete disruption. Zenker and von Ziemssen[1] quoted Boerhaave's[2] description in 1724 of the necropsy on Admiral Baron Wassenaer, wherein Boerhaave mentioned the "esophagus, whose continuity had been sundered, and the two portions had been dragged respectively upward and downward."

Barrett,[3] writing in March, 1946, stated that more than fifty cases of spontaneous rupture of the esophagus had been recorded since Boerhaave's case and that in none of these cases had treatment been successful. In August, 1946, Eliason and Welty[4] made a similar observation qualified by mention of two cases of Graham in which the rupture resulted in empyema and the patient recovered after drainage of the empyema.

Walker[5] in 1914 suggested that, if diagnosis was made early, the patient might be saved by posterior mediastinotomy, the rent being repaired and the mediastinum and pleural cavity drained. Shock and sepsis made him think success would be unlikely, but in the past one and one-half years much has been accomplished by surgical attack along these lines. A complete study of the subject, with report of five cases, in one of which the patient underwent operation but died nine days postoperatively of a pulmonary embolism, will soon be published by Kinsella, Morse, and Hertzog.[6]

Also, one of us (T.O.C.) has recently performed repair successfully in one case of this so-called spontaneous rupture of the esophagus.

Much speculation has gone on concerning the status of the esophagus prior to "spontaneous" rupture. It is thought that despite the lack of ulcer, stricture, neoplasm, or other visible lesion the esophageal wall cannot be healthy if it becomes the site of a rupture. Zenker and von Ziemssen explained this by esophagomalacia—a weakness inherent in the tissues. The case reported here is not one of those in which rupture occurs in an otherwise normal esophagus. Our patient had a congenitally short esophagus type of hiatal hernia, which almost always has associated with it a stricture at the esophagogastric junction.

That esophageal strictures predispose to, or at least often precede, rupture of the esophagus is suggested by the reports of Vinson,[9] Smead,[7] Weder,[8] and others, who, however, described lye strictures,[9, 10] usually 3 to 5 inches (8 to 13 cm.) above the cardia. In these cases, rupture occurred between strictures and cardia owing to the unrelieved or slowly relieved pressure of vomiting.

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The present case is unusual in several respects, but mainly because this is a case of total or complete disruption, occurring at the site of stricture.

REPORT OF CASE

The patient was a widow, aged 54 years, who had had no previous illness except typhoid fever in 1897. She was referred to the Mayo Clinic by her local physician, who had noted albuminuria and pyuria for the previous eight months and had made a presumptive diagnosis of a ureteral stone. She had had no renal colic, hematuria, or chills and fever. She had lost from eight to ten pounds (3.6 to 4.5 kilograms). She complained, in addition, of some pain and soreness of the left foot which she had felt for the previous three to four months.

Physical examination showed a small woman walking with a painful limp of the left lower extremity. There were no masses or tenderness in the abdomen. However, there was questionable tenderness in the left costovertebral angle. The arch of the left foot was tender. Laboratory examinations revealed albuminuria, grade 3 (on the basis of 1 to 4, in which 1 designates the mildest and 4 the most severe condition), and pyuria, grade 1. Roentgenograms of the left foot showed osteoporosis and a fracture of the left third metatarsal bone. Roentgenograms of the thorax showed slight osteoporosis of the ribs. Investigation of the kidneys, ureters, and bladder revealed a stone in the lower part of the right ureter and operation was advised. Hyperparathyroidism was considered to be likely but not to contraindicate operation.

On July 28, 1947, one of the urologic surgeons explored the right ureter and removed a stone from the intramural portion of the ureter. Before operation the patient had vomited slightly but had had no other gastrointestinal symptoms. During operation, which was done while she was under spinal anesthesia, she vomited rather violently but this was not considered unusual in view of the anesthesia. Early the following morning, July 29, 1947, the patient vomited again. This time there was old blood in the vomitus. In addition, difficulty in breathing began to develop. As it was thought that there had been aspiration of some of the vomitus, an attempt was made to aspirate the stomach. However, the tube could not be introduced into the stomach. A roentgenogram of the thorax revealed mediastinal emphysema and an aberrant position of the end of the stomach tube, apparently not in the esophagus and certainly not in the stomach. The patient was seen by one of the consultants on diseases of the thorax, who noted subcutaneous emphysema of the thoracic wall and neck. These findings, together with the roentgenographic appearance of the thorax and the degree of shock in which she was, led to the probable diagnosis of rupture of the esophagus and mediastinitis. The possibility of a previous asymptomatic esophageal hiatal hernia or of an epiphrenic diverticulum was entertained.

When we saw the patient it was felt that further diagnostic studies would be both impossible and wasteful of time in view of her condition, and accordingly, exploration of the inferoposterior mediastinum was done through a vertical incision parallel to the lateral edge of the left sacrospinalis muscle at the level of the eighth and ninth ribs. Posterior segments of these ribs were resected as well as the intervening intercostal muscle bundle. Extrapleural dissection into the posterior mediastinum revealed discolored, edematous, gangrenous tissues and with final exposure of the esophagus and stomach, there was revealed a complete disruption of the esophagus from the stomach. Approximately 2 to 3 inches (5 to 8 cm.) of the stomach was in the thorax (congenitally short esophagus type of hiatal hernia). The two vagus nerves were the only connections remaining between the esophagus and the stomach. The ends of these latter two viscera were inflamed, friable, and necrotic. In addition, there were openings into both pleural spaces which made the maintenance of anesthesia and respiration extremely difficult. It was impossible to consider an anastomosis of the esophagus to the stomach, and the patient was returned to her room, where she died approximately twelve hours later.

Post-mortem examination confirmed the operative findings described in the preceding paragraph and in addition confirmed the preoperative suspicion of hyperparathyroidism by

the finding of multiple parathyroid adenomas. An incidental finding was a small islet-cell carcinoma of the pancreas which had metastasized to the duodenum and to the peripancreatic and choledochal lymph nodes.

COMMENT

Several interesting possibilities arise in one's mind concerning the cause of, or the predisposing factors in, this disruption of the esophagus from the stomach.

Certainly it seems, first of all, that a congenitally short esophagus type of diaphragmatic hernia, with its well-known concomitant stricture at the esophagogastric junction, would definitely predispose mechanically toward such a catastrophe. If, in addition, the disturbance of calcium metabolism and the presence of a carcinoma with metastasis (asymptomatic) lent a systemic effect deleterious to strength and elasticity of tissue it seems easy to understand how an attack of violent vomiting was all that was needed to effect the disruption.

The fact that this was a *total disruption* seems best explained on the basis of the pre-existing lesion. The stricture almost always accompanying congenitally short esophagus is of the complete circumference of the esophagogastric junction. However, there are many patients encountered with such lesions, and undoubtedly many of them occasionally vomit; yet there is no known previous instance of disruption among this group as far as we can determine, although rupture of the esophagus occurs fairly often in patients with strictures from other causes.

In the case reported here the prognosis was completely hopeless. The duration of the rupture was probably twelve to fifteen hours and the general condition of the patient, the local status of the mediastinal tissues and ends of the esophagus and stomach, and the presence of bilateral hydropneumothorax all combined to preclude any possibility of operation.

Had the diagnosis been made earlier it is conceivable that an anastomosis might have been possible, the necrotic ends of the two separated viscera being resected if necessary. Early diagnosis, however, requires early consideration of the possibility of rupture of the esophagus, and as yet this fortunately rare catastrophe is not commonly borne in mind.

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J. E. H. Roberts

NOTICE

The twenty-ninth annual meeting of the American Association for Thoracic Surgery will be held on March 29, 30, and 31, 1949, in New Orleans, La. Headquarters will be at the Roosevelt Hotel.

Requests for hotel reservations should be mailed direct to Dr. Alton Ochsner, 1430 Tulane Avenue, New Orleans 13, La.

Abstracts for presentation of papers for the 1949 meeting must be received for consideration by the Program Committee, on or before Jan. 1, 1949. These abstracts should be submitted in *quadruplicate* and addressed to the Secretary of the Association, Dr. Brian Blades, George Washington University Hospital, 901 23rd St., N.W., Washington 7, D. C. They should not be sent to the Editor. The early date of the 1949 meeting makes it absolutely necessary that these abstracts be received before the deadline date. Abstracts received after Jan. 1, 1949, will not be considered.

Manuscripts for competition in the Rose Lampert Graff Prize should be sent directly to the Secretary of the Association not later than Jan. 1, 1949.

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